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No. 6 FEBRUARY 8th, 1958 Vol. 70

Registered for Transmission as a Newspaper *Price 2s. post free*

Bovine Mastitis

IN spite of constant additions to our knowledge of the problem, and in spite of an ever-increasing array of therapeutic agents, bovine mastitis still continually faces the veterinary surgeon in large-animal practice. The enormous economic loss resulting from disease of the udder in dairy cattle has been made abundantly clear by a series of surveys and calculations. Thus, the Economic Advisory Council Committee on Cattle Diseases, in its 1934 report, drew attention to the fact that in surveys undertaken at three different centres and extending over periods of one to three years, the disposal from herds due to udder disease amounted on average to no less than 7.7 per cent. (with a range of 4.2 per cent. to 15.0 per cent.). In 1940, the Survey Committee of the then National Veterinary Medical Association at a conservative estimate put the annual financial loss to the dairy industry resulting from decreased milk production associated with mastitis at £3,000,000. This figure was based on a price of 1s. 3d. per gallon of milk. Much more recently, Withers (1957) has recorded that in a survey undertaken in herds in Berkshire, Surrey, and Wiltshire, the case incidence of mastitis was 10.2 per cent. for 1952-3 and 14.5 per cent. for 1953-4. The 1956-7 report of the Production Division of the Milk Marketing Board, referring to data collected from the field, states that "It was found that, on any day in the year, on average about one cow in every 100 cows in milk has a hard quarter or clots in the milk." These are all exceedingly important figures for an industry constantly striving for higher productivity in face of ever-mounting costs.

By all who are actively interested in the problem, whether as veterinary surgeons in practice or as research workers, it is readily accepted that there are many factors, infective and otherwise, which can contribute to the bovine mastitis syndrome. Each case, or at any rate each herd problem, has to be treated on its merits.

Some of the infective agents, notably *Streptococcus agalactiae*, can be dealt with effectively by most available antibiotics, others are less sensitive.

Some of the environmental factors involved are now better understood and can be corrected. On the other hand, this aspect of the problem still calls for much more experimental work

It is as well to remember that great progress has been made in tackling bovine mastitis, and that the clinician to-day is in a far better position to deal with most forms of the disease than was the case as recently as 20 years ago. Not only is there a much fuller appreciation of the problem itself, but there are now available greatly superior therapeutic agents.

In order to place before our readers a summary of the latest information concerned with bovine mastitis, the Technical Development Committee has enlisted the aid of several workers in this field to contribute to the current series of refresher articles. The first of these contributions, "The Progressive Pathology of Bacterial Mastitis," appears on another page of this issue of THE VETERINARY RECORD. It is hoped to follow this in the next two weeks with two further papers entitled "Mastitis Caused by Bacterial Infection," and "Factors Predisposing to Mastitis with Special Reference to Milking Technique." These have been written by research workers. The fourth paper in this series, "The Clinician's Approach to Mastitis," will take the form of a summing-up by a practising veterinary surgeon.

Refresher Article No. 2

The Progressive Pathology of Bacterial Mastitis

BY

I. H. PATTISON

Agricultural Research Council, Field Station, Compton, Berks.

Introduction

THE terminal pathology of bovine mastitis is well documented. Indeed, Helmboldt, Jungherr, and Plastridge (1953) have noted that in 1886 Kitt considered that nothing could be added to what was then known about the pathology of mastitis. The excellent paper by Helmboldt *et al.* has summarised the pathological investigations that have been made on this condition and has described the pathology of mastitis associated with streptococci, staphylococci, corynebacteria, and certain other organisms. Stamp (1943), and Huddleson (1934) have described, respectively, the pathology of tuberculous mastitis and of mastitis caused by *Brucella abortus*.

It is well recognised, then, that the end result of bacterial invasion of the mammary gland may be involution, fibrosis, necrosis, or gangrene. However, the progressive pathological processes involved in the production of these end-products are less well understood. How, why, and how quickly do these changes take place, and what factors control the duration and severity of the inflammatory reaction in any individual gland?

This paper summarises the work on the progressive pathology of experimental streptococcal and staphylococcal mastitis that has been carried out at Compton, and emphasises those observations that may be of practical significance to clinicians.

Streptococcal Mastitis

From experimental observation, it seems likely that the way in which *Str. agalactiae* and *Str. dysgalactiae* cause mastitis is similar to that of most organisms which enter the mammary gland through the teat canal, but that the degree of tissue damage depends on the virulence of the invading organism. By virulence is meant "disease-producing potential," a mixture of toxicity and invasiveness. It is felt, therefore, that a description of the mechanism by which *Str. agalactiae* causes mastitis will explain much that occurs in other bacterial forms of the disease.

Infection of the cow with *Str. agalactiae* takes place only through the teat canal. The number of organisms required to establish infection experimentally depends on the resistance of each individual animal, the nature of which is not fully understood, but in certain circumstances, that are probably associated with the invasiveness of the organism and the susceptibility of the animal at any particular time, the organism multiplies rapidly within the milk ducts and initiates what we at Compton have called a "crisis." Each crisis is a sequence of events that gives rise to the classical clinical picture of streptococcal mastitis, and an understanding of the crisis explains the progressive pathology of the disease.

The crisis syndrome. The crisis syndrome is shown diagrammatically in Fig. 1. The essential mechanism of each crisis is as follows. There is a sudden multiplication of organisms within the milk-ducts measured by a sharp rise in the bacterial count of the milk. The organisms pass through the duct walls into the adjacent lymphatic vessels, and this stimulates an immediate defensive outpouring of neutrophils from the capillary blood vessels to the milk-ducts, measured as a sharp rise in the neutrophil content of the milk. The animal's temperature rises suddenly, and there are the usual clinical signs of fever. Bacteriological culture of the supramammary lymph node at the time of crisis is frequently positive, in contrast to negative culture at all other times, and an increase in mouse-lethal power has been demonstrated in streptococci isolated from the milk at this time when compared with the culture originally inoculated, or with cultures obtained from the milk at other times. Histologically, in the mammary gland of animals killed during a crisis, organisms can be seen passing through the duct epithelium into the lymphatic vessels, a majority being immediately phagocytosed by defending cells. In both the goat and the cow each crisis is characteristically brief, usually lasting 12 to 24 hours.

These crises are almost invariably limited to the first month after experimental infection, and a succession of crises indicates progressive invasion of fresh areas of udder tissue—for tissue damage in streptococcal mastitis is typically patchy: one area may be unaffected, while an adjacent area is rendered completely functionless.

The progressive pathology of streptococcal mastitis. At the time of movement of streptococci through the duct walls a great neutrophil response is noted (Fig. 2*). The presence of invading cocci calls for cellular defence, with neutrophils and macrophages crowding into the area (Fig. 3) and the duct epithelium reacts by becoming hyperplastic (Fig. 4). Invasion through the duct wall is patchy and the reactive hyperplasia is patchy also (Fig. 5). By using the inert, easily identified, acid-fast organism *M. phlei*, plus hyaluronidase to give it penetrating power, Howell (1957), working in this laboratory, demonstrated conclusively the patchy nature of bacterial penetration from the milk-ducts to the underlying tissue, and the corresponding localised reaction to the invading organism.

Once a tissue reaction has been set in motion it will continue until the irritant has been subdued or

* This and subsequent Figures appear in the centre pages of this issue.

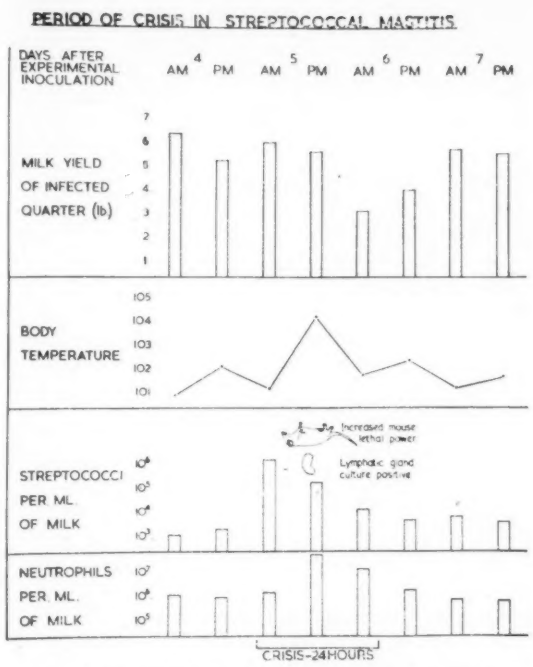
PERIOD OF CRISIS IN STREPTOCOCCAL MASTITIS
DAYS AFTER EXPERIMENTAL INOCULATION
AM 4 PM AM 5 PM AM 6 PM AM 7 PM
MILK YIELD OF INFECTED QUARTER (lb)
BODY TEMPERATURE
STREPTOCOCCI PER ML. OF MILK
Increased mouse lethal power
Lymphatic gland culture positive
NEUTROPHILS PER ML. OF MILK
CRISIS-24 HOURS

FIG. 1.—A period of crisis in streptococcal mastitis.

eliminated. The response to a low-grade pathogen such as *Str. agalactiae* involves first the neutrophil, then the macrophage, then the fibroblast. Damage to the duct wall, especially of the small secreting ductules and acini, interrupts milk secretion. If these various processes are added together, the proceeding stages of the disease are readily understood. Interruption of milk secretion indicates damage to secretory epithelium and fluid stasis in the secretory acini (Fig. 6). Macrophage and fibroblast activity indicates increased cellularity of interalveolar tissue (Fig. 7), progressing, in places, to frank fibrosis (Fig. 8). A mammary gland that is prevented from secreting milk begins to involute, in the same way as it does at the end of each lactation; the acini collapse, and the lobules shrink (Fig. 9). Thus, there remains a partially fibrosed, largely involuted gland, reduced in volume and yielding only a fraction of the milk produced before the streptococcal invasion began.

From the clinical point of view it is noteworthy that the swollen, painful gland that is often called "fibrosed" is not really fibrosed but indurated with retained secretion and tender inflammatory tissue. The truly fibrosed gland is small and pliable on palpation; large areas of such a gland are functionless.

Changes in the ducts are produced by the invading cocci. If the duct is large the reaction takes the form of epithelial hyperplasia, and if the irritation is prolonged the hyperplastic epithelium takes on a pavement character and may even acquire a cornified surface layer (Fig. 10). If the duct is small a different change occurs due to pressure from the surrounding tissues; at the point of penetration of the cocci the epithelium and the cells underlying it begin to multiply *into* the duct, forming an invagination of the wall (Fig. 11); this invagination of granulation tissue with an epithelial covering enlarges very rapidly and may eventually block the duct completely (Figs. 12 and 13.)

As the acute stage of streptococcal mastitis passes, the hyperplastic duct epithelium and the intraductal invaginations break down and are passed from the udder as "clots" in the milk. The damaged areas of the gland are functionless because of fibrosis or involution, and the ducts become patent again and regain their normal epithelial lining. (Fig. 14).

Staphylococcal Mastitis

The staphylococcus is potentially a more dangerous organism than *Str. agalactiae*. It is a toxin-producer—sometimes to a marked degree—and many strains elaborate enzymes that add to their pathogenicity. It follows that defence against staphylococci is likely to be more difficult than against streptococci, and that with some strains tissue damage will be more extensive and more serious.

Studies begun by Derbyshire (1957a) on experimental staphylococcal mastitis in goats have shown 3 distinct reactions to experimental infection with the same strain of staphylococcus inoculated through the teat canal, (a) gangrenous mastitis, (b) severe non-gangrenous mastitis, (c) mild mastitis. These reactions were not strictly related to the number of organisms injected, although large numbers usually produced an extensive reaction. Clinical observations and examination of the blood picture and bacterial and neutrophil content of the milk suggested that the first stage of the disease was essentially the same as in streptococcal mastitis, namely multiplication of the organism within the milk-ducts and invasion through the duct wall to the interacinar tissue. In mild reactions further progress was similar to that in streptococcal mastitis, but in severe and gangrenous reactions it was obvious that much more serious damage had occurred than has been encountered in streptococcal infections. There was evidence that the crisis syndrome sometimes occurred in staphylococcal mastitis, but that in the gangrenous and severe non-gangrenous forms of the disease the organisms usually established themselves in the udder tissue in one massive initial invasion.

The progressive pathology of staphylococcal mastitis. The complete series of histopathological changes which occur in staphylococcal mastitis has not yet been worked out, but sufficient is known (Derbyshire, 1957b) to indicate important differences between streptococcal and staphylococcal mastitis. In severe staphylococcal mastitis the histopathological changes are much more marked, due to the fact that staphylococci, unlike streptococci, can live and multiply outside the milk-duct system. The extent of the initial invasion of udder tissue from the ducts may well be similar in both diseases, but the staphylococci are not completely removed by phagocytic cells and establish themselves in numerous foci where they multiply and cause extensive damage in their

immediate vicinity (Fig. 15). Histological examination indicates that foci containing staphylococci may originate either intraductally or extraductally anywhere in the udder. The defence reaction to staphylococci is intense, and protective cells crowd into the area obliterating the mammary gland structure (Fig. 16). Walling off of the irritant area with fibrous tissue occurs very rapidly (Fig. 17). In areas of the gland not actually invaded by staphylococci, involution associated with duct blockage and milk stasis may occur. In spite of the large numbers of living staphylococci throughout the udder tissue, there is no evidence that they enter the general circulation, although, from time to time, they may reach the supramammary lymphatic gland.

The progress of the disease from this acute stage will depend on the amount of mammary tissue involved and the success with which the invading organisms are eliminated. If infection is widespread large areas of the gland will be destroyed; if, however, the infective foci are few in number and are rapidly walled off the effects will be less serious. In any event there will be fibrosis and involution, loss of milk, and shrinkage of the quarter.

In gangrenous staphylococcal mastitis there is marked vascular congestion together with oedema, that may be caused by thrombosis of the main veins draining the udder, associated with acute inflammation in the adjacent mammary tissue. Gangrenous changes may be a hazard in any acute case of staphylococcal mastitis, and can occur early in the disease and progress rapidly; the dark blue colour spreads visibly over the skin in a few hours, and the gland becomes cold and tense; the udder secretion is watery and blood-stained. There is no evidence that the presence of bacteria additional to staphylococci is necessary to cause gangrene. Gangrene is an irreversible, pathological change, the tissue involved being completely destroyed, and it is accompanied by a generalised toxaemia which may be fatal.

Corynebacterial Mastitis

No study has yet been made of the progressive pathology of corynebacterial mastitis. The various tissue changes which end in the extensive necrosis characteristic of the disease may only be imperfectly deduced, therefore, by comparison with the changes that precede necrosis in acute staphylococcal mastitis. But it seems likely that the two processes are similar, and that each advances rapidly. The observation of Helmboldt *et al.*, that in corynebacterial mastitis the ducts are extensively lined with epithelium similar to the stratified squamous epithelium of the oral cavity, suggests that the invasion of corynebacteria from the ducts to the underlying tissue is generalised throughout the affected quarter. Such a massive invasion would, of course, increase the severity of the initial stages of the disease.

Mastitis Associated with Other Organisms

In Britain, "bacterial mastitis" usually signifies mastitis caused by streptococci, staphylococci, or corynebacteria. From time to time, however, cases of bacterial mastitis associated with other organisms are encountered. Virtually nothing is known of the progressive pathology of these forms of mastitis, and it may be that certain variations in the general pattern exist. For example, Helmboldt *et al.* have indicated that "lymphocytic-type" cells rather than neutrophils predominate in coliform mastitis.

The Relationship of Progressive Pathology to Treatment and Prognosis

Streptococcal mastitis. Antibiotic treatment *via* the teat canal usually eliminates *Str. agalactiae* from an udder and if this treatment is given early in the disease the prognosis regarding future milk production is good. There appear to be 3 reasons for this. Firstly, the organism is sensitive to appropriate antibiotics. Secondly, it remains alive only in the milk ducts where it can be reached by the drug, and is rapidly destroyed by the body defences during penetration from the ducts at a time of crisis. Thirdly, it is a low-grade pathogen that does no more than stimulate epithelial hyperplasia, fibrosis, and involution. The considerable speed with which pathological changes take place indicates that treatment should be prompt, otherwise the chances of duct-blockage that may hinder penetration of the antibiotic are increased. If the animal is dull in addition to having a swollen quarter, the indications are that a crisis has occurred and that the disease is progressing rapidly. Since for the efficient treatment of streptococcal mastitis the requirement is a sufficient concentration of antibiotic throughout the milk duct system, there does not appear to be any reasonable basis for injecting antibiotics by any route other than the teat canal.

Occasional cases of *Str. agalactiae* mastitis are very resistant to antibiotic treatment, although the organism is sensitive when tested *in vitro*. The obvious inference is that the drug is not reaching the organism within the udder. I have examined histologically a few udders of this kind, and have noted that occasional cocci were apparently embedded between cells of the duct epithelium; in this position they may have been protected from a lethal concentration of the drug. It is not clear whether this unusual type of case is related to some peculiarity of the organism, or of the host, but from a practical point of view it would seem reasonable to remove cows of this kind from the herd.

Prognosis regarding milk production from a quarter damaged by streptococcal mastitis depends on the ratio of fibrosed to involuted tissue within the gland, and on the stage of lactation of the animal. If the streptococci have been eliminated early in the disease from a gland in early lactation, it is likely that the milk yield will gradually rise again and may reach the pre-infection level. If, however, treatment has been delayed and lactation is advanced, the affected quarter is quite likely to dry off completely. There is no obvious reason why involuted tissue should not lactate again in the following lactation, and, again, compensation for fibrosed mammary tissue may occur through development of new secretory tissue. All these variations in local response to

streptococcal damage have been observed experimentally, but it is not always easy to predict milking potential for each individual case. The general principle is that early and successful treatment of a case in early lactation is likely to be followed by a return to near-normal milk flow; prognosis in other cases will be relative.

Staphylococcal mastitis. Treatment of staphylococcal mastitis is often difficult, and prognosis uncertain, the main reasons being that some staphylococci are resistant to antibiotics, and that it is often difficult to bring the antibiotic into contact with the staphylococci. The pathological processes occurring within the udder cause duct-blockage, and the multiplying organisms may be protected by necrotic and fibrous tissue through which the drug will not diffuse. The blood supply to necrotic areas is interrupted, and this reduces the likelihood that antibiotic injected by routes other than the teat canal will reach the staphylococci. In the earliest stages of acute cases, however, parenteral medication may successfully bring the antibiotic into contact with the invading organism.

When the virulence of the organism is high, the initial invasion of mammary tissue from the milk ducts following experimental inoculation of a large number of staphylococci through the teat canal causes fever, dullness, and local tenderness of the udder in 4 to 6 hours, and the animal may die of gangrenous mastitis in less than 48 hours. This period of dullness, produced experimentally, probably corresponds to the time at which a farmer would seek assistance, and it is important, therefore, to deal with such cases as a matter of urgency. If treatment is carried out before duct-blockage and localisation of the staphylococci in inaccessible parts of the gland occur, the antibiotic has a reasonable chance of reaching the invading organism. When gangrenous changes are recognised clinically it would appear unlikely that treatment with antibiotics can save the quarter.

Prognosis in staphylococcal mastitis is difficult and should be guarded until the initial response to treatment has been observed. Early treatment in early lactation warrants the most favourable prognosis. Any marked systemic disturbance indicates considerable reduction in milk yield.

Acknowledgment.—Part of the work described in this paper was originally published in *The Journal of Comparative Pathology and Therapeutics* (1951, 61, 71; 1952, 62, 1), and I am grateful to the editors of that journal for permission to reprint some of the illustrations.

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Kerato-conjunctivitis in Broiler Birds

BY

C. N. SAUNDERS

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SUMMARY.—An outbreak of kerato-conjunctivitis in broilers is described.

A small-scale experiment successfully reproduced the disease with ammonia but failed to show any transmissible infective agent.

It is concluded that exposure to ammonia arising from the litter is directly concerned with its causation.

Introduction

THE first description of kerato-conjunctivitis in poultry was given by Bullis, Snoeyenbos & Van Rockel (1950) who reported that the condition was first noticed in the U.S.A. in 1943. Experimental work on its aetiology was carried out by Faddoul & Ringrose (1950), who suggested that ammonia liberated in the litter was concerned in its causation. More recently, Wright & Frank (1957) in Canada reached a similar conclusion.

There is no published account of its occurrence in Great Britain, although Venn (1957) has encountered a similar condition. This paper records details of an outbreak seen in Huntingdonshire, together with the results of an attempt to reproduce the disease experimentally.

Details of Field Outbreak

Housing and Management

A total of 30,000 birds was kept in units of 1,000 to 2,000 in ex-War-Department brick buildings.

The outbreak occurred in one house of dimensions 70 feet long by 30 feet wide, 10 feet to the eaves and 16 feet to the apex of the roof, which held 2,200 birds. A central staging at a height of 2 feet with infra-red lamps suspended above at intervals provided the brooding area where the whole batch of day-old chicks was confined. Later, the full run of the floor was given, and a trough space of 3 inches per bird allowed. No injuries were known to have occurred from overcrowding.

Fourteen louvre windows on each side of the house provided cross ventilation and there was also one extractor fan in the roof which was in constant operation. Despite this, however, an uncomfortable smell of ammonia was present. The litter consisted of wood shavings and in the house concerned had been used from October, 1956 until April, 1957, having carried two previous batches of birds. It appeared to be in fair condition, being neither dusty nor wet. The normal practice of the owner was to discard all damp and caked litter and to heap the remainder for about a week to heat, after each batch of birds. New litter was then spread under the staging anll 2 feet on each side of it, for the chicks. As the fud area of the house came into use the old litter was spread out. The litter in each house was completely

renewed each year. The litter used for this particular batch had not, however, been treated as efficiently as usual after the last group of birds.

The owner had experienced a similar outbreak of disease the first time he had re-used litter, when he was less experienced in its management and when it was definitely in poor condition. That outbreak occurred in October. The outbreak now described occurred in April. On both occasions ventilation was restricted, particularly at night.

The ration fed in pellet form was of a consistency slightly more crumbly than usual. There were no hanging sacks, wires or other physical hazards present. The windows were permanently reddened, allowing only a dim light.

Clinical Signs

Symptoms were first noticed when the birds were 6 weeks old, and a week later it was estimated that about 60 were affected (approximately 3 per cent.). By the 14th day 20 more had developed lesions, but the earlier cases were beginning to recover. The birds were slaughtered at 11 weeks old by which time the eyes of most of the birds were almost normal.

Affected birds sat quietly in the dark corners of the house, with their eyes closed. Some were seen to rub their eyes with their wings. When disturbed the affected birds were as lively as the others, but when handled their bodily condition was found to be poorer.

In the majority of cases both eyes were equally affected, but in a few there were unilateral or unequal lesions.

The lesions consisted of a single shallow erosion of the cornea, with a clearly demarcated irregular edge. Most of the lesions were central, but a few were slightly posterior to central. They varied in size from 1 to 5 mm. in diameter. The unaffected area of the cornea was bright and normal in appearance and the ulcerated area only very slightly opaque.

In early cases there was slight conjunctivitis which became more marked and was accompanied by oedema after one week. When the eyelids were forcibly retracted in bright light excessive lachrymation occurred. The iris was normal in colour and outline.

Diagnostic Investigations

Post-mortem examination of several affected birds revealed no evidence of intercurrent disease. Since avitaminosis A is commonly associated with affections of the cornea an examination of liver vitamin A levels was made which showed satisfactory values ranging from 1,014 to 1,370 I.U. per gramme. Microscop cally the corneal lesion was seen to be a desquamation and erosion of the surface epithelium. There was no evidence of invasion by bacteria or fungi nor were any other tissue reactions noted.

No inclusion bodies or rickettsia were seen in direct smears stained by Giemsa and modified Ziehl-Neelsen, and cultures on blood agar incubated in air for 48 hours ei her remained sterile or yielded sparse growths of non-haemolytic streptococci only.

Inoculation of the chorioallantois of 14-day chick embryos with an emulsion of corneal lesions in saline gave negative results. Two out of the 7 embryos died but they, with the remainder examined after 7 days, showed no evidence of infection.

Transmission Experiment

In an effort to determine whether an infective agent was present a small transmission experiment was undertaken. Transmission was attempted by two means :—

1. By instillation into the left conjunctival sac only, on two occasions with an interval of 4 days, of finely triturated affected cornea suspended in saline.

2. By co-habitation of one affected and two healthy birds.

Since previous work (Faddoul & Ringrose, 1950) had suggested that ammonia played an important part, it was arranged that the birds should be divided into two lots, one half being maintained in an atmosphere charged with ammonia.

Thus 4 experimental groups were made up as follows, the results being shown alongside :—

Groups	Results
A. INSTILLATION *Normal atmosphere* Two—4-week-old growers	No eye lesions after 49 days.
B. INSTILLATION *Ammonia atmosphere* Two—4-week-old growers	Developed lesions between 43 and 48 days.
Six—2-week-old chicks	Four developed lesions between 33 and 48 days. Two died. No lesions.
Two—2-week-old chicks Non-instilled to act as controls.	Died between 35 and 38 days No lesions.
C. CONTACT *Normal atmosphere* Two—4-week-old growers with one affected bird	No eye lesions after 49 days.
D. CONTACT *Ammonia atmosphere* Two—4-week-old growers with one affected bird.	Died after 10 days. No lesions.

Of the birds maintained in an atmosphere of ammonia 6 out of 12 developed lesions between the 33rd and 48th day after exposure, whereas all the birds maintained in a normal atmosphere (Groups A and C) whether instilled or not, remained unaffected until the end of the experiment after 7 weeks. It will be seen that the atmosphere containing ammonia, rather than the instillation, was the dominant feature. The naturally affected donor bird recovered spontaneously and no eye lesion was visible after 3 weeks.

The atmosphere of ammonia was obtained by allowing approximately 150 ml. per day of 0.88 ammonia solution to drip into a tray at ground level in a closed room of 414 cubic feet. This atmosphere

was slightly uncomfortable to the human eyes and nose. At the end of 7 weeks the birds were unaffected and the ammonia concentration was then doubled. It was now distinctly irritating and, after 12 days of this higher concentration, or 33 days total exposure the first eye lesions were noted.

Four of the birds in Group B died in the next few days with signs of respiratory distress and *post-mortem* lesions of tracheitis. The non-instilled controls were among these casualties and they failed to develop eye lesions.

All the birds in Group B which survived the experiment eventually developed bi-lateral lesions simulating the natural condition. It is significant that, although the left eye only was used for instillation, lesions appeared equally in both eyes with no preference for the instilled eye. Group D however suffered a mishap and all the birds died after 10 days through other causes, without any lesions having developed.

The surviving birds were examined 7 weeks after the beginning of the experiment. *Post-mortem* examination showed no evidence of intercurrent disease, and Vitamin A liver levels were normal.

The growth of those birds exposed to ammonia was much reduced in the later stages, their mean bodyweight being 760 grammes at 11 weeks of age, compared with 1,047 grammes for the birds in a normal atmosphere. Anorexia was a marked feature after eye lesions had developed.

Discussion

Other workers have failed in attempts to transmit the condition (Bullis *et al.*, 1951; Wright & Frank, 1957). The results of this experiment, though limited in scope, agree with their results. It has however been produced by maintaining birds in ammonia atmosphere only, without any attempt at transmission (Faddoul & Ringrose, 1950; Wright & Frank, 1957).

The condition has also developed when birds were deliberately housed in unhygienic conditions for other experimental purposes (Barber, 1947). The chief factors concerned appear to be :

(i) Ammonia production consequent upon the re-use of litter.

(ii) Inadequate ventilation resulting in the stagnation of ammonia-laden air, in particular under the brooder baffles. In this connexion it is noteworthy that the condition is mostly confined to the winter season.

(iii) Floor brooding, the birds thus being in constant proximity to the source of gaseous decomposition.

In the present case a " high energy " (5.5 per cent. fat) ration was being fed, but Bullis (1950) states that outbreaks have been observed where both high fat and normal rations were in use, and discounts the importance of the former.

Two breeds (L.S. × R.I.R. and Barred Rock × R.I.R.) were equally affected in this outbreak and " 404 " hybrids were used in the experiment.

Other writers make no special mention of the breed involved and it would appear that there is no particular breed susceptibility.

The importance of kerato-conjunctivitis lies in its effect on the growth rate. The profit margin in broiler production is now so narrow that any check in thriving is of considerable economic importance. Prevention should therefore be the aim and it is suggested that this may be achieved by close attention to the ventilation, particularly in the winter months, and careful treatment of the litter which is intended for re-use.

(This paper was received for publication on November 26th, 1957.)

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FIFTY YEARS AGO

Our great predecessor, William Hunting, appears to have been in vigorous form 50 years ago. His leading article, of which the opening is quoted below, is in entertaining contrast to the manner of THE RECORD in these days. He wrote:—

" Last week we printed a communication from Mr. J. A. Dollar, which showed that that gentleman, with many expressions of humility, had entered upon a somewhat belated opposition to the new Act. Since then Mr. Dollar has favoured the profession with a circular couched in terms anything but humble, and accompanied by a stamped post-card. It is possible that some unthinking members, misled by Mr. Dollar's wealth of extravagant verbosity, have already answered his appeal as he desires. Our advice to all others is—put the post-card in the fire.

" We cannot analyse this extraordinary manifesto in detail. We have explained and approved the principle of the annual registration scheme so often already, that it seems superfluous to dwell upon Mr. Dollar's absurd ideas regarding this main point. There are minor points regarding which it would be easy to show the fallacy of his arguments—if it were worth while. But the fact is that the circular so teems with misrepresentations and exaggerations, and affords such frequent evidence of the writer's lack of knowledge of the subject, that it scarcely deserves notice at all.

" Mr. Dollar, a member of Council and an ex-President of the profession, really knows less about the Bill than does many an obscure practitioner. He attended the last Council meeting—after a year's absence—to oppose the Bill, evidently not knowing that it had already gone to the Privy Council. Had he chosen to read the report of the October Council meeting he would have known—as everyone taking more than a spasmodic interest in the matter knows—that the Council then adopted the Bill, and deputed a Special Committee to present it to the Privy Council without delay."

Experiences with Cage-birds

BY

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SUMMARY.—A survey of 463 cases of disease in 435 cage- and other pet birds is presented.

A form of classification has been attempted in order to assess the relative importance of the conditions.

The aetiology, nature, diagnosis, treatment, and outcome are discussed and summarised where possible and the paper concludes with notes on the general handling of these birds.

Introduction

DURING the past 25 years the cage-bird population of this country has considerably increased. This is mainly due to the great increase in the popularity of budgerigars for whereas in the early years of the century canaries formed the bulk of the bird pets, to-day budgerigars overwhelmingly predominate.

In the past, veterinary surgeons, with one or two outstanding exceptions, have taken little interest in the diseases of cage-birds and the treatment of these creatures was largely in the hands of breeders who by their observations amassed a considerable amount of useful knowledge, empirical though much of it must have been.

Perusal of available scientific literature indicates that interest in cage-birds has been confined chiefly to three channels: firstly, a purely genetical one largely in colour inheritance; secondly, the endemic and epidemic features of the "French Moult" syndrome, both of which have been studied by eminent scientists, notably in Germany and the United States and to a lesser extent in Austria, France, and Britain; and thirdly, a group of somewhat sketchy references to a heterogeneous collection of disease conditions based largely on analogy to the domestic fowl in some instances and in others to wild birds and pigeons.

TABLE I

CAGE AND OTHER PET BIRDS ATTENDED

Budgerigars	358
(Males 172, Females 89, Sex indeterminate or not recorded 107)	
Canaries	39
Pigeons	27
Parrots	13

and 1 each of Finch, Linnet, Blackbird, Pope Cardinal, Red Cardinal, Java Sparrow, Love-bird and Macaw.

TABLE II

GENERAL CLASSIFICATION OF CONDITIONS MET

	Budgerigar	Canary	Pigeon	Others	Total
Injuries					
A. Fractures	35	3	6	1	45
B. Burns	10	1	1	1	13
C. Other injuries	58	2	6	1	67
Tumours	54	1	1	1	57
Dermoid Cysts	12	10	–	–	22
Abscesses	9	–	–	–	9
Beak Troubles	49	–	–	1	50
Ectoparasites	31	5	1	–	37
Feathering and skin disorders	26	6	–	1	33
Digestive disorders	22	1	4	4	31
Respiratory disorders	17	3	5	3	28
Nervous disorders	28	2	1	2	33
Reproductive disorders	6	–	–	–	6
Developmental abnormalities	6	–	–	1	7
Unclassified	27	1	1	2	31
Total conditions ...					463

Material Studied

The present communication relates to 435 cage-birds and other birds presented for treatment at the Veterinary Hospital of the University of Liverpool during the 2½ years 1955 and 1957. Some 40 others which simply required their claws cutting are omitted.

This disease picture is influenced by the fact that the cases dealt with were almost entirely pets, generally with no contact with other birds. Thus, the opportunity to study epidemiology was very limited. Similarly isolated cases of suspected nutritional disorders could not be positively diagnosed.

Injuries

Fractures

Six birds sustained fractures on the pulleys of the rope-suspended type of clothes rack. The fractures were usually compound and sometimes multiple with gross contusion. Shutting the perching bird in a door accounted for 4 similar fractures of leg or legs. Wounds of the leg below the identity rings were accompanied in 6 cases by fracture and the ligature effect of the ring on the swollen tissues caused necrosis and dry gangrene in three. One pigeon was presented with wing fractures due to gunshot pellets. The remaining fractures were of unknown causation. The commonest site was the tibio-tarsus (23) followed by the tarso-metatarsal region (6). In 4 there was tarsal fracture/dislocation. Four humeral fractures, 4 femoral (one of these being a fracture dislocation of the hip), and one digital fracture completed the list.

Even in the most severely contused cases, including those where necrosis and gangrene had set in, shock and malaise appeared slight and brief. The time occupied in actual repair was about half that noted in the dog or cat.

In the early cases of the series the only treatment applied in simple fractures was to place the bird in

a smooth-walled box. Repair occurred rapidly but alignment varied and subsequent serviceability was poor in the majority of cases, the deformed limb being an encumbrance. Firm callus formed in 6 to 10 days in most cases. Later it was decided, after reduction of the fracture, to secure the limb in a resting position in the case of the wing and in the perching position when a leg was involved, by plastering or trussing. Adhesive and plaster of paris bandages were used for this purpose. This positioning was found to be important as it greatly influenced the speed and degree of rehabilitation. If the bird was tolerant of this restriction, the dressings were left in place for one to two weeks. Feet were either left uncovered or enclosed with the digits only slightly curled as if grasping a thick perch. It was not found possible to immobilise midshaft and high femoral fractures by these methods and such cases were left unplastered.

With wing fractures, the bird always held the affected limb low and unless it was raised and supported, drooping deformity resulted. Restrictive, elastic plaster supports encircling the body were applied at three levels, *viz.* firstly thoracic, secondly abdominal, and thirdly wing and tail feathers. This treatment at first interfered markedly with balance and sometimes frightened the bird. Wild birds with wing fractures brought to the clinic by kindly members of the public were destroyed, because it was thought that such birds would not be able to compete successfully with normal birds or evade their enemies.

Compound fractures were reduced by traction. In some cases where bone was still projecting through the skin, it was necessary to remove a little of the shaft to effect reduction. The wound was then dressed with a dusting powder, usually a mixture of penicillin and sulphadimidine or sulphanilamide, and left uncovered for two to four days. By this time, continuity of the skin was regained and the fracture was treated as a simple one.

In 3 birds, leg damage was so gross that necrosis had commenced. In these, the flaccid limb was ligated with an encircling ligature and the dead tissue cut away. The birds showed no resentment. In 2 other cases, although on removal of the ring the limb seemed healthy, it had sloughed away within 7 days. In none of these birds were there any signs of toxaemia or other systemic disturbance.

As to the outcome, it was found that in birds which had had their legs in semi-flexion with open claws during healing there was a more rapid and more complete return to function than those in which the limbs had been immobilised in the fully flexed or fully extended positions. Three of the five wing fracture cases were known to fly again but did so in a rather lop-sided and weakly manner. Flaccid paralysis remained in a few of the high leg fracture cases, but with the majority of lower lesion cases, good use was subsequently made of the limb.

Burns and Scalds

Burns. All the recorded cases met resulted from flying into the fire and only the legs appeared to suffer permanent damage. Presented birds showed varying degrees of damage from singeing and distortion of a claw to the complete absence of both feet; the last creature being obliged to hobble on its hocks. Again, the birds showed little signs of pain or shock, and if not extensively damaged, perched well.

Treatment consisted of painting the affected areas with 1 per cent. gentian violet solution and the subsequent application of a bland ointment daily, although recovery was virtually just as satisfactory without treatment. The outcome was usually favourable, and provided that most of the digits remained, walking and successful perching was possible, even by a bird having short, clawless stumps on all its toes.

Scalds. Three budgerigars scalded by hot water or fat showed a greater degree of shock. Feathers of the legs, breast, and abdomen fell out within 48 hours leaving a marked erythema and some irritation. Treatment was similar to that used for burns.

Other Injuries

The most important of these related to a tourniquet-effect of leg-rings and it is hoped that an expanding ring or a wing tag will be increasingly used and recognised by breeders to obviate this unnecessary and often cruel sequel to use of the rigid rings.

In 4 birds brought for *post-mortem* examination, deep puncture wounds believed to have been inflicted by a cat's claw or teeth were found. Another 4 birds showed little more than mild shock after being known to have been in cats' or dogs' mouths. Pecking by cage mates or wild birds was the suggested cause in at least 5 cases of head injury, including 2 pigeon squabs. Head injuries were commoner in adipose or weakly birds. The remaining group included several accidental injuries, and it would seem that allowing these pets to fly about the average living-room is fraught with danger. The great activity of the birds contributes to the frequency of injury to the limbs in the wires of the cage or its baubles. This has accounted for several cases in continually caged birds.

A two-year-old racing pigeon showed a swelling over the left clavicular region with a sinus leading into its centre which contained a mass of cheesy material from which a coliform organism in pure culture was obtained. It was learned that several months previously the bird had returned from a flight with gunshot wounds and crop contents visible in the region of the lesion. Dissection revealed another sinus in the right clavicular region. It appeared that the exudate had distended the interclavicular air sac. Despite the mass of material the bird had recently won races.

As a rule, wounds healed very rapidly and suppuration was not a feature even in the most contused and lacerated types. Moreover, with few exceptions, interference with wounds by the bird itself was negligible. When the wound was severe a disc type of Elizabethan collar, made from thin cardboard, was fitted as a precaution. Where birds shared living quarters, isolation was the first step to prevent

further mutilation. With ringed birds the ring was removed immediately when injury or slight swelling was detected below the ring, using claw clippers. Removing a tightly embedded ring was often a difficult and bloody operation and unless done early, slough distal to the ring resulted. Antibiotic creams with surface anaesthetics incorporated were used in a few cases where discomfort and erythema were marked. Varied diets containing animal protein foods—milk, eggs, finely minced meat, and liver—and fruit plus green vegetables, in addition to the seed mixture were advised. Powdered yeast and cod-liver oil were also recommended in severe cases. Relative rest in a smooth-walled box with a glass top appeared to speed recovery.

Tumours

Under the term "tumour" is included a variety of local growths of differing aetiology, site, and structure. The majority were associated with the subcutaneous tissues, but a considerable number of abdominal, thoracic, and skeletal new-growths are included. Histological examination indicated some striking differences from mammalian tumours. Tumour sizes varied from a few millimetres to over two centimetres in diameter, and in several, liquefactive and fatty necrosis and myxomatous and hyaline changes were present making classification difficult. In some, consequent on their size and position, flying and perching were interfered with, while in others, pressure symptoms—respiratory and circulatory embarrassment or collapse—and alimentary disturbances, were noted.

Of the benign tumours, fibromata and papillomata were conspicuously absent; the commonest single condition being that of a lipomatous type in the sternal region. Of such cases which were subsequently available for *post-mortem* examination, most showed an abnormally heavy distribution of depot fat and had fatty hearts and livers. These birds felt appreciably heavier than normal on handling and in some this increased weight was so marked that flight was impossible. There is little doubt that the relative inactivity of cage life, coupled with a diet rich in seeds containing a high unsaturated fat level such as is found in the cheaper seed mixtures, is the chief cause of the condition. In the larger sternal lesions, areas resembling the fat necrosis of the brisket of the ox, and myxomatous changes were present. In two, hyaline areas containing sparsely distributed cells were reminiscent of an abnormal type of cartilage. One budgerigar showed at *post-mortem* examination a coiled, membranous structure, like a watch spring and covered by a fold of peritoneum in the anterior abdomen. Histology showed it to be hyaline in structure with here and there calcium deposits. In a bird with an ovarian neoplasm, an area of endochondral ossification was also present in a swelling over the lumbo-sacral vertebral mass. A small number of myxomata, with or without an admixture of cystic spaces, fat or blood clot, were encountered, mostly situated near the crop. One heavy bird was presented with a large fluctuating abdominal swelling which on incision proved to be a rupture of the abdominal muscles. At necropsy, in addition to excessive fat distribution, including in the liver, a granulomatous lesion was found at the rupture site with giant cells and crypts which in life had contained cholesterol crystals. In one bird showing opisthotonus, gasping respirations and a bias to the left, subsequent autopsy showed a very large cyst of the liver with yellow serous contents. In a 5-year-old male budgerigar, in addition to a fibrinous peritonitis, there was present a mass the size of a pigeon's egg which proved to be granulomatous rather than neoplastic, and although various giant cells were seen, stained sections failed to reveal acid-fast organisms. The lesion was in intimate contact with a necrotic hepatic cyst.

Among the classifiable malignant tumours, carcinomatous and sarcomatous types were of equal frequency, and together their number almost equalled that of the classifiable benign growths. The term "types" is used because of some doubt as to whether strict analogy with mammalian histology is applicable and whether they were true neoplasms.

Six testicular and two ovarian neoplasms, the former generally resembling seminomata, one of which was a Sertoli-cell tumour, were seen, and the main and sometimes the only symptom was respiratory embarrassment. One or both organs were involved and the largest was 2.5 by 2 by 1 cm. Metastases in the liver were found in one case. Adenocarcinoma of the liver was encountered in a 3½-year-old canary.

One case was seen in which a tumour of the loin and prefemoral regions proved to be dermal cyst but with further epithelial development indicating malignancy. Histologically parts resembled a basal cell carcinoma. This was the only skin lesion which bore any resemblance to an epithelial neoplasm and it suggests that prolonged irritation of an abnormal epithelial structure may provoke malignant changes.

Three tumours of the wing were found to be of the sarcoma type. The tissue of origin was uncertain but bone marrow was involved and several types of cell varying from ill-defined spindle cells to plump oval ones with many mitotic figures were observed. One of these cases was a definite osteogenic sarcoma of the humerus with rarefaction and enlargement and a metastatic lesion which occupied one-third of the thorax. One bird with a protuberance of the breast showed mild myeloid leucaemia in addition to a myxosarcoma of the breast and lungs. It is interesting to record that this bird had had a sarcomatous growth removed from the under surface of the wing 6 months previously. In several subcutaneous tumours, dry or liquefactive necrosis had occurred which on bacteriological and histological examination could not be distinguished from chronic abscesses.

Surgical interferences of considerable magnitude were endured by budgerigars with little sign of operative shock. Metastasis seldom occurred before the primary neoplasm was easily discernible and therefore operative removal was usually attempted. Intra-abdominal neoplasms were generally inoperable. Even in the conscious bird, little resentment was shown to incision, except incision about the head and when blunt dissection was resorted to. Consequently,

anaesthesia was seldom called for. Ether inhalation was found to be the safest method of anaesthetisation and in 3 cases was given to a depth of full anaesthesia; in others light narcosis only was induced with ether. Chloroform was found to be invariably fatal within a minute of inhalation. Effective anaesthesia was produced by the intraperitoneal injection of pentobarbitone sodium in doses of 1/20th to 1/40th grain in healthy budgerigars, but the same dosage was fatal in seriously ill ones.

Surgical methods employed were as follows: unless very large, ill-defined, or very vascular, incision was made and ligation, forci-pressure, and dissection effected a complete removal. The more difficult lesions were incised, biopsy material taken, and if not highly malignant or invasive in type, controlled destruction by phenol or a similar agent applied on pledgelets of cotton wool was carried out at weekly intervals. In this way, haemorrhage was controlled, progressive necrosis produced, and when the lesion had become more circumscribed, the remainder was extirpated by dissection. All surgical wounds, even the intra-abdominal ones, were dusted with penicillin and sulphadimidine powder and sutured with fine gut. Healing was rapid and there was no tendency for the birds to interfere with the wounds. These methods were used with success in most of the benign and a few of the sarcoma-type lesions. It is now realised that with the lipomatous group of cases, any symptoms exhibited were more likely the result of liver changes than of the growth itself, and surgery is useless.

Dermal Cysts

In this group of 22 cases the equal number of canaries and budgerigars involved suggests a greater proneness to the condition in the former for the ratio of totals of the two breeds in this survey is 1:9.2.

In appearance the lesions which have been likened to the pilonidal sinus of man and other mammals, were growths much like a neoplasm. They were rounded, with or without peduncle, sometimes multiple (in one canary distributed over the entire surface of the body) and occurred in a wide range of sites, being commonest on wings, less common in the cloacal region, breast, and dorsum of the body, and occasionally on the head and legs. Ulceration occurred in some cases but irritation was not a feature. When ulcerated, the yellow, granular, and characteristically musty and sebaceous-smelling contents, bulged through the acorn-cup-like, fleshy cyst wall. The contents were examined microscopically in the dry state and in 10 per cent. KOH, revealing epithelial scales, sebaceous material, free fat, and various immature stages of feather development. Unopened cysts were usually sterile or only contained saprophytic bacteria. Occasionally anthracoids or non-haemolytic staphylococci were isolated. Culture and microscopical examination for fungi proved negative and no arthropod or other parasites have been found. In non-ulcerating cases differentiation from true neoplasms and from abscesses can only be made by incision and examination of the contents, and in some cases even with careful histological and bacteriological examination it is not always possible to differentiate.

Effectively to deal with the lesions it was necessary to extirpate completely the cyst contents and lining membrane, or where possible, the entire cyst. In large sessile or diffuse lesions it was found that incision, curettage, and destruction of the lining membrane with a corrosive was most satisfactory. Phenol proved both effective and safe and was applied on a minute pledgelet of cotton wool to the cyst lining and adherent debris. When necessary this process was repeated at weekly intervals, curettage of black necrotic material allowing the fleshy wall gradually to contract and thus lessen the vascularity. After each cauterisation, the excess phenol was wiped away and sulphanilamide or other dry dressing dusted into the cavity. The greatest danger was haemorrhage, and this was the limiting factor to surgery. Stage-by-stage removal was the only feasible method in many cases. With pedunculated lesions, an encircling ligature around the stalk, or simple crushing of the stalk with Spencer-Wells forceps achieved good haemostasis, and the mass distal to the forceps or ligature was scraped or cut away.

When extirpation was incomplete a recurrent lesion was present in a few weeks. One lesion only showed signs of malignancy in the form of basal cell development in some areas with numerous mitoses.

Abscesses

Nine is an unexpectedly small figure for these lesions and all except 2 were on the scaled parts of the legs. Three were multiple cases in the form of yellow pustules. Only 2 cases were examined bacteriologically and a Pasteurella and *Staphylococcus aureus* produced the great bulk of colonies on culture.

Abnormalities of the Beak and Related Structures

All but one parrot were budgerigars and ranged from 6 weeks to 5 years of age. Two-thirds of the cases were of overgrown upper beak, often with a shallower curvature than normal. On trimming to normal proportions, the corium was found to be longer than normal and appeared to extend with successive clippings. Eight cases of cross or wry beak were presented, also overgrown in some degree, and a clinical cnemidocoptic infection was present in 5 of them. One bird had a thickened, overgrown and rubbery upper beak, and it was learned that the bird had recently been in lay. A case of complete fracture and loss of the upper beak was seen. During the succeeding 4 months, beak horn grew to a length of 7.5 mm., beyond which there was no development in spite of clipping back the lower beak. Appetite and feeding were unimpaired.

It is fairly certain that at least a proportion of the beak deformities had their origin in nestling infestations by Cnemidocoptes spp. and the resulting inflammation produced irreparable changes in the generative tissue. Insufficient opportunity for wear seems to account for a few cases of overgrown beak, and the suggestion that it is due to lack of calcium or vitamin D in the diet is not convincing. Uneven

wear or chipping of the beak can initiate wry-beak at any age.

Overgrown beaks were trimmed with the standard nail or claw clippers. Regrowth invariably occurred though the rate varied greatly. Bleeding points were at first treated with an antiseptic powder. Later thermo and chemical cauteries were used in an attempt to limit the terminal horn production, but without result. Early wry-beak was checked by repeated trimmings but complete correction has not been obtained in the small number treated.

A condition, not found mentioned in the literature, was found in an advanced state in 12 budgerigars. The name tentatively given here as "Brown Hypertrophy of the Cere" is fairly descriptive. The cornified waxy surface of the cere progressively thickens and turns dark brown in colour and the external nares become surrounded by a high wall anteriorly. In mild cases in males the owner often assumes sex-reversal to have occurred. In females, the slow stratification of brown material is often passed unnoticed. Bacteriological investigations have shown no recognised pathogenic bacteria. One or two yielded anthrocoids of unlikely pathogenicity and common contaminants. No fungi or arthropods were found, although one bird subsequently developed "scaly leg." Microscopical investigation of the horny but friable material showed in the main, greatly stratified epidermal cells with some sebaceous material.

In so far as treatment is concerned, no success has been achieved with common antiseptics and antibiotic ointments, penetrating agents such as gentian violet and alkyl ammonium compounds, or acaricides like mesulphen.* The horny material could fairly easily be chipped or scraped away, enlarging the external nares where these were blocked and revealing the normal cere colour beneath. Nothing, however, prevented recurrence in a few weeks. Dietary variations and vitamin supplements were prescribed without appreciable effect.

Ectoparasites

Only one disease condition has been recognised with certainty under this head. This is known variously as "Scaly Face," "Scaly Beak," or "Scaly Leg," according as to the prominent symptom, and is well recognised by breeders.

The predilection structures were attacked in any order in a freshly infested bird, and the frequency was roughly in the following order: upon the cere, over the upper beak, around the eyes, and lastly on the scaled parts of the legs. Ages affected were from 6 weeks to 5 years.

The palpebral and cere lesions developed as amber- or beige-coloured flakes of dried granular exudate and epithelial scales. On the beak, infestation often appeared as a "bloom," which when examined by hand-lens showed as a beige honeycomb with minute, contiguous burrows riddling its surface. Pruritus was often absent and never a marked feature. Occasionally some encroachment was seen on to the feathered parts of the head, the commissures of the beaks, the lower beak, and the fine, featherless skin behind the lower beak. With leg lesions, the scales of much of the shank and digits were raised by similar, but more chalk-like, deposits which were most exaggerated over the limb flexures and here the pressure and friction of walking and perching movements sometimes produced bleeding. Leg movements were not appreciably restricted and lameness was slight.

The condition was not simulated by other unrelated conditions and the clinical appearance alone was found sufficient to warrant acaricidal treatment. To confirm the diagnosis, liberal scrapings of the lesions, especially the honeycomb beak lesions, were taken. Maceration in 10 per cent. KOH and mounting in rape oil were both used; the former in an attempt to reveal the causal mite, and the latter to show clearly the burrows. The burrows which are some 250 to 350μ in diameter, were easily demonstrated under a 2/3 objective, but female and larval mites were only demonstrated in 12 head and 2 leg cases. Of these, three were positively identified as *Cnemidocoptes pilae* (Griffiths & Lavoipierre), a recently identified species. The mite is more fragile than *C. mutans* and maceration must be carefully controlled. Mites *in situ* in burrows were difficult to distinguish when mounted in oil. Identification as

* Odylen, Bayer.

The Progressive Pathology of Bacterial Mastitis.—*Concluded.*

FIG. 2.—Streptococcal mastitis: the first reaction to bacterial invasion of the udder: neutrophils passing from capillaries through the wall of a secretory acinus.

FIG. 3.—Streptococcal mastitis: an early stage in bacterial invasion: neutrophils and macrophages congregating under slightly hyperplastic duct epithelium.

FIG. 4.—Streptococcal mastitis: a later stage in bacterial invasion: macrophages have replaced neutrophils and the duct epithelium is clearly hyperplastic.

FIG. 5.—Streptococcal mastitis: patchy hyperplasia of duct epithelium.

FIG. 6.—Streptococcal mastitis: fluid stasis in secretory acini and damage to secretory epithelium.

FIG. 7.—Streptococcal mastitis: marked increase in the cellularity of inter-acinar tissue.

FIG. 8.—Streptococcal mastitis: fibrosis, one of the terminal stages of the disease.

FIG. 9.—Streptococcal mastitis: involution, the other terminal stage of the disease.

FIG. 10.—Streptococcal mastitis: stratified, cornified epithelium lining a large duct.

FIG. 11.—Streptococcal mastitis: an invagination of granulation tissue into a small duct, the invagination occurring at the point of penetration of the cocci.

FIG. 12.—Streptococcal mastitis: a mass of granulation tissue, originating from an invagination (as in Fig. 11), completely blocking a duct.

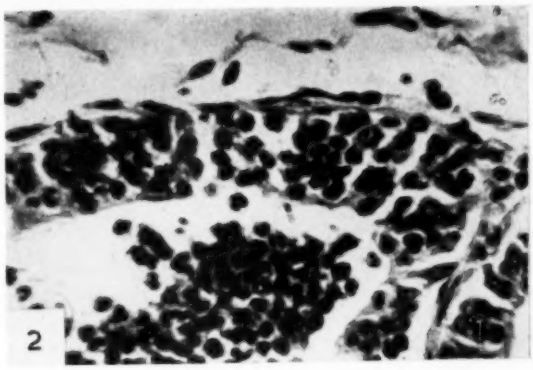
FIG. 13.—Streptococcal mastitis: low-power view of three ducts partially or completely blocked by granulation tissue and cell debris. Note that the adjacent lobular tissue is normal, confirming the patchy distribution of lesions.

FIG. 14.—Streptococcal mastitis: the appearance of a large duct in the terminal stage of the disease when all evidence of epithelial hyperplasia has disappeared.

FIG. 15.—Staphylococcal mastitis: darkly-staining foci of staphylococci surrounded by necrosed tissue.

FIG. 16.—Staphylococcal mastitis: extensive necrosis and obliteration of gland structure.

FIG. 17.—Staphylococcal mastitis: a thick wall of fibrous tissue separating an area damaged by staphylococci from normal gland tissue.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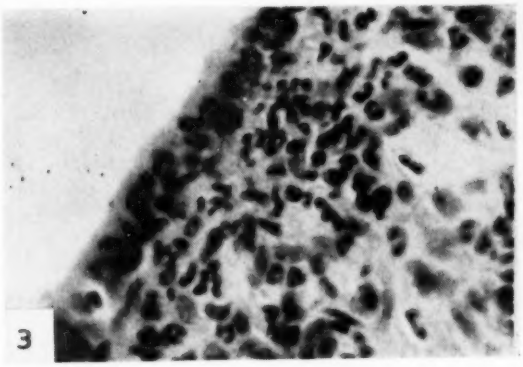
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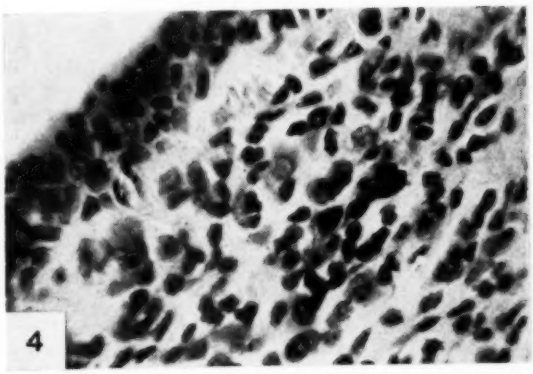
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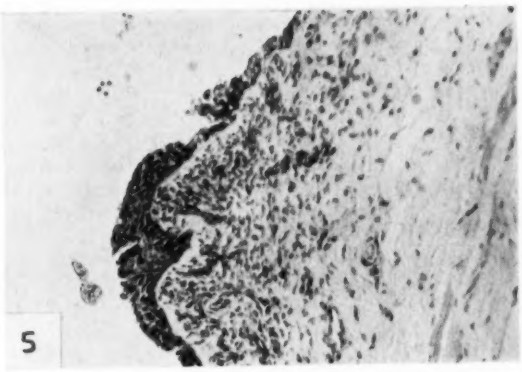
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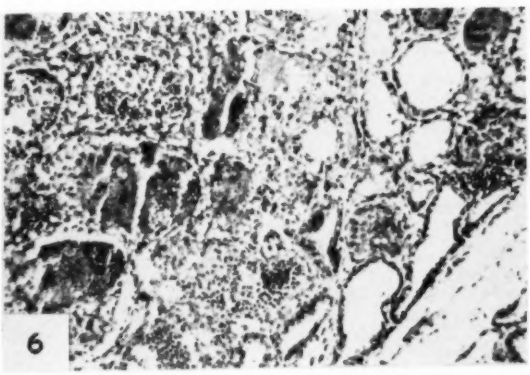
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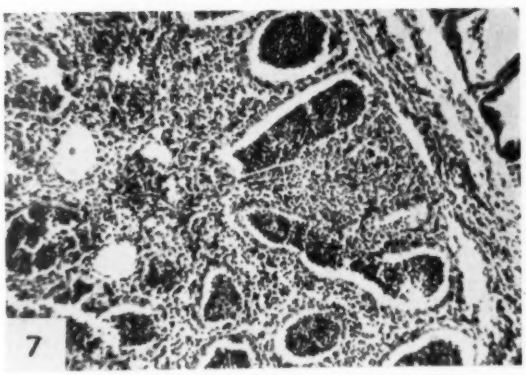
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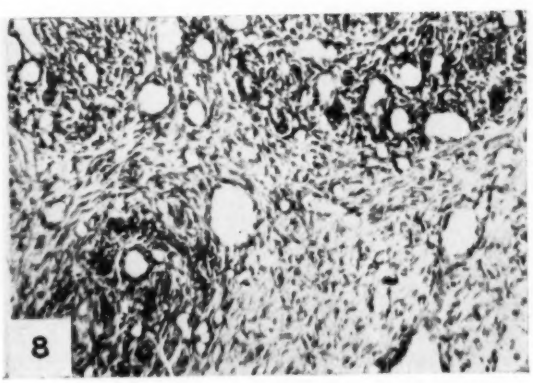
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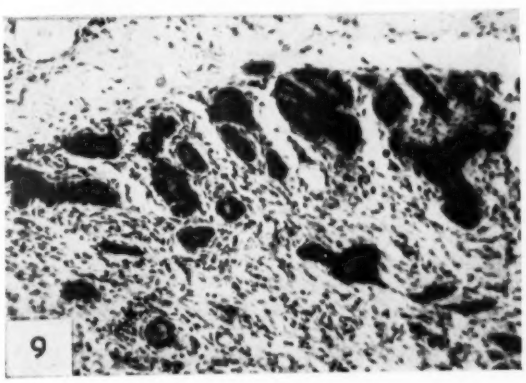
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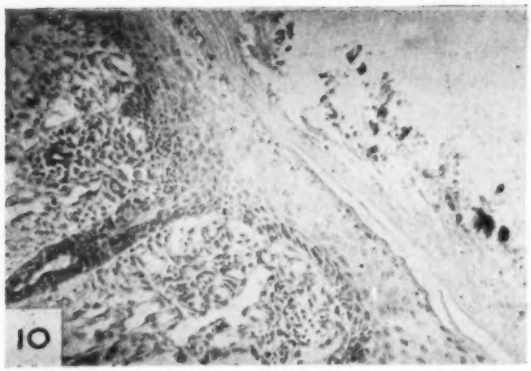
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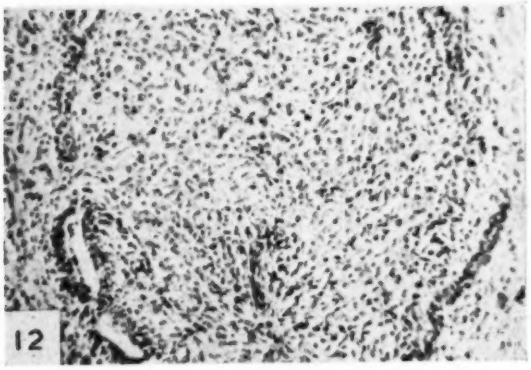
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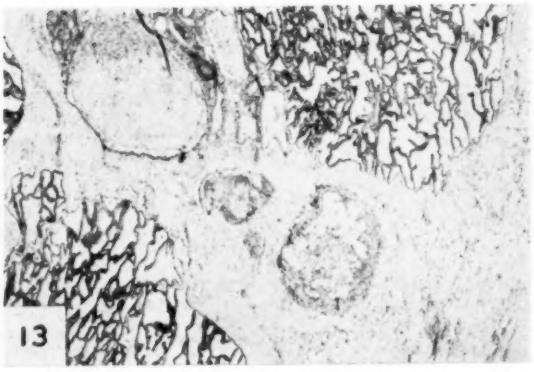
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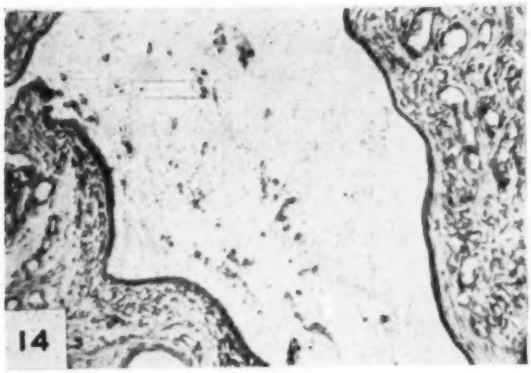
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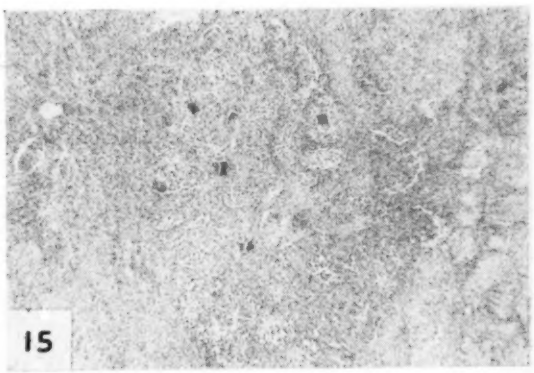
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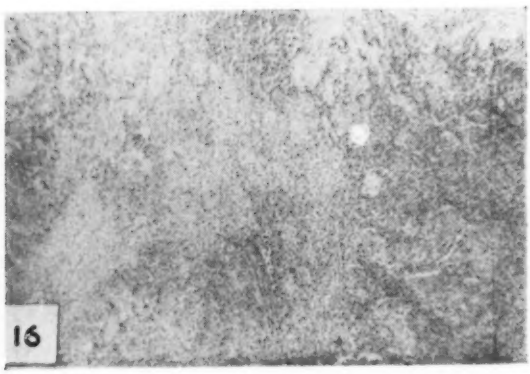
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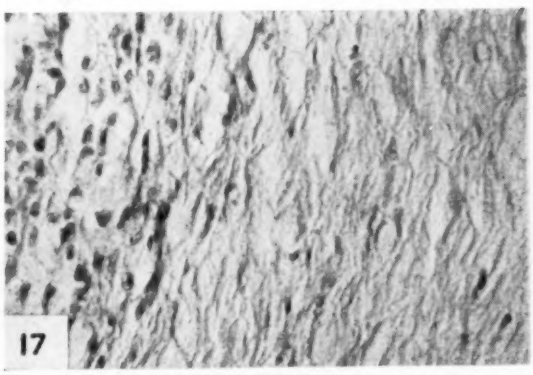
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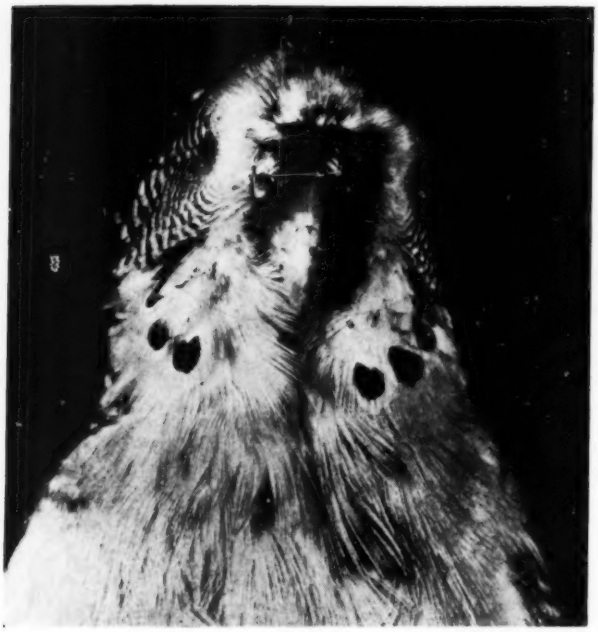
A., B. and C. show Budgerigar beak deformities resulting from long-standing Cnemidocoptic infestation, usually acquired during the nestling stage.

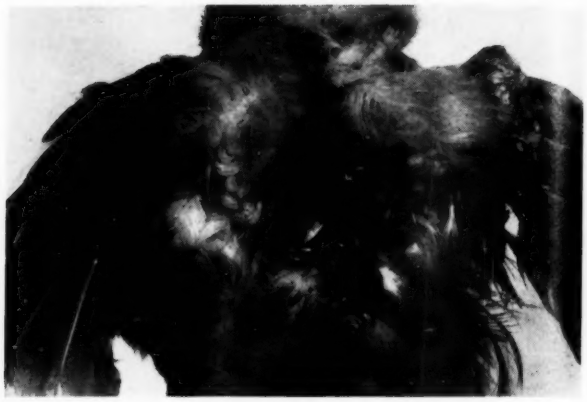
A.—Enlarged plate showing individual burrows of the Cnemidocoptes on beak, cere and eyelid, and flaking of the superficial layers of abnormal horn. Both beaks enlarged, thickened, friable, irregularly worn and referred to in the text as " Shovel Beak."

B.—Showing a denser, more spatulate beak deformity with little residual evidence of mite infestation.

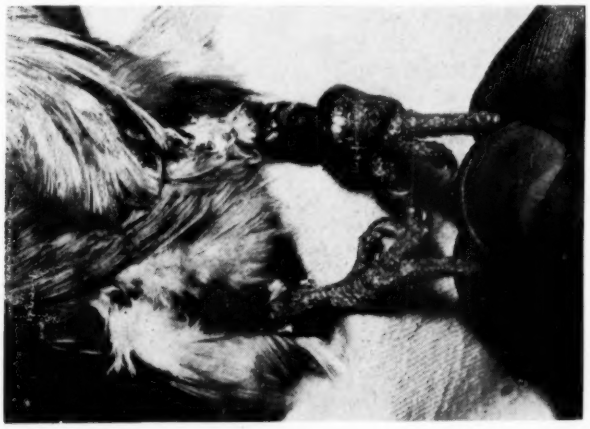
C.—Unusual case of overgrown upturned beak. After treating the infestation, and with regular trimming of the beak, this bird

D.—Canary, two years, showing bilateral herniation of the clavicular air sac, in which, on incision of the right lobe, was seen a pulsating venous sinus, 5 mm. in diameter. This case was presented subsequent to the survey period and is not men-



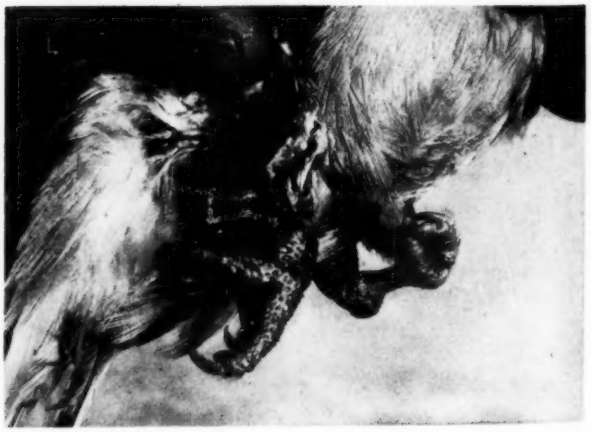


Multiple diffuse dermal cysts in a five-year-old canary, some of which are ulcerated and show curled and deformed feather tufts, associated with the lesions. This bird was destroyed on grounds of humanity.



Views of a case of brown hypertrophy associated in this case with excessive depot and organ fat, and a lipomatous mass on the breast.

The condition seems to be associated with several chronic conditions, and may simply be a symptom of general organic deterioration.



Advanced case of oedema of the lower right metatarsus, photographed as soon as the cause, a metal ring tag, had been removed. Some power eventually returned to the limb, but the claws remained flaccid. Congenital or nestling hock deformity of the left leg, which in a younger bird might have been mistakenly diagnosed as perosis, was found upon the bird's death some weeks later to yield a pure culture of a Lancefield's Group G. streptococcus, which may have entered the body through the ring lesion where the organism was also found at autopsy. The bird was three years old.

a Cnemidocoptes species is not difficult but exact identification requires an experienced taxonomist.

It is interesting that a Cnemidocoptes species was identified, though not typed, from a scraping of the wing of a homing pigeon with local covert feather loss. In no other bird in this survey has feather loss been associated with Cnemidocoptes, nor indeed with any other mites, except a possible Dermanyssus infestation. A condition of thickened leg scales in 5 canaries suggested a parasitic aetiology but scrapings proved negative for mites, although 2 birds recovered clinically on 2 occasions, one year apart, after courses of painting the lesions with tetraethylthiuram monosulphide.* In 2 canaries so affected, toes dropped off in handling to obtain scrapings. The impression created by these and subsequent canaries is that the condition occurs mainly in older birds, and may have a physiological basis.

For Cnemidocoptic infestation two treatments were used: mesulphen† 60 per cent. in rape oil at strengths of from 1:3 to 1:1 was applied with a child's paint-brush every 2nd or 3rd day for 3 or 4 weeks, after as much as possible of the mite debris had been scraped away. Similarly tetraethylthiuram monosulphide 25 per cent. w/w at strengths of 1:29 to 1:9 in water or 70 per cent. alcohol was used. Alcohol maintains solution of the latter but cannot safely be used near eyes or external nares. Except in very early, mild cases, little response was noted before 3 to 4 weeks, when the exudative lesions began to crumble and fall away. Three to 12 weeks elapsed before normal appearance was restored but if treatment was applied efficiently the condition seldom recurred. Only 2 cases recurred again at least 6 months after apparent recovery.

Toxicity was not noted in any bird treated by these two drugs even after 6-week courses of treatment, but owners were advised of possible toxicity and to use sparingly, avoiding the eyes, nares, and tongue.

In very advanced cases and particularly in nestling stage infestation, thickening, fragility, lateral deviation, "shovel-beak," overgrowth and irregularity of growth of beaks have been noted, some permanent damage having occurred in the horn-producing organ. In two cases of advanced scaly leg, digits were lost, apparently due to vascular restriction by the hampering, concreted scales and debris.

It is not understood why an isolated cage-bird should suddenly develop a flourishing mite burden unless a subclinical infestation has been present from the nestling stage. Questioning of owners failed in most cases to show any recent contact with other cage-birds, wild birds, second-hand fomites, or having visited a pet shop or similar bird handler. A possible contact is with food bought from the pet shop and like places, but this presupposes a considerable resistance by the mite or its larvae to temperature and humidity changes and to prolonged separation from the host. Visits to premises of dealers in cage-birds showed several young budgerigars with clinically apparent scaly face on sale to the public.

In 4 cases a horn-like structure had developed below the eyes and near the temporal canthus. They were some 4 mm. long by 2 mm. diameter. Pathological examination of these structures showed them to consist entirely of necrotic epithelial scales, fat, and debris. No bacteria of significance were found. The suggestion that Cnemidocoptes might be involved was not borne out by the investigations although one of the birds subsequently developed typical "Scaly Leg." The condition is well recognised by breeders and their treatment consists of repeated paintings with 2 per cent. liquor cresolis saponatus (Lysol), but the success and toxic hazards of this are not known. In the 3 cases recorded the lesions were brushed or scraped off and bleeding points painted with 1 per cent. gentian violet. No bird was returned with regrowth.

Feathering Disorders

This is a broad group in which any irregularity of the plumage such as abnormal growth, self-interference, abnormal loss and poor appearance and wearing qualities has been included. In only one case, a homing pigeon, was a parasitic cause demonstrable. All others were of unproved aetiology, although one budgerigar was clinically and by history a classical French Moult "hopper." This was the only case seen of this much discussed but little understood condition. In 6 cases a possible Red Mite (Dermanyssus) infection was suspected, but in only 2 had owners seen a "red sand" on the perch and in the crevices of the cage. Neither was able to produce a specimen of the "sand."

Postulated causes of feather loss, poor feathering, and pruritus are many. They include painful trauma, overheating, variable environmental temperature, poor ventilation, boredom, fear (*e.g.* marauding cats), over-petting, nervous and physical exhaustion, over-long hours of natural or artificial light, monotonous, stale, or unbalanced diet including low or poor protein foods, absence of green food, absence of shell and grit, and the inevitable vitamin panorama. Additionally, where more than one bird is kept in a cage or sun parlour, persecution by the stronger bird may be added to the list. Symptoms varied from a small area of erythema and feather loss or continuous slight feather fall, to complete baldness and vain attempts at tufty feather growth. A small proportion showed marked irritation. In view of the multitude of possible causes of irregularities of plumage, the treatment adopted while speculative was as comprehensive as possible. Dietary changes were frequently suggested. Variation and the following additions were favoured: powdered yeast, strictly limited cod-liver oil, fresh green food including several garden weeds, ripe fruit in small quantities, occasional replacement of milk for water, and very finely minced meat and liver. Occasionally aneurin hydrochloride and biotin were prescribed. It was felt that the accepted all-grain diet was not entirely appropriate for psittacines at least, partly because of its excess of carbohydrates,

* Tetmosol.

† Odylen.

and more particularly the high content of unsaturated, fatty substances, for the relatively inactive life the birds lead. Evidence for this has become more abundant in recent months with an increasing number of cases of fatty liver, fatty heart, and excessive depot-fat deposition.

In those cases where an intermittent parasite such as a Dermanyssus mite was even remotely suspected it was recommended that the cage be scrubbed with hot washing soda water and the perches and all crevices be painted with a nicotine sulphate wash or acaricide suspension. As continuous parasitism was not suspected, spraying or dusting of the actual bird was not considered necessary or advisable.

Self damage was uncommon and even where present an Elizabethan collar of thin cardboard was preferred to risking matting the feathers with an ointment, as this encouraged interference and excessive preening by the bird.

Digestive Disorders

The group includes conditions in which the main symptoms were relatable to the alimentary tract and subsidiary digestive dysfunctions. The most frequent type met in budgerigars has been regurgitation from the crop of food, gas, froth, or discoloured fluids. One budgerigar and one pigeon had crops impacted with dry, concreted material. Coprophagia was noted in some of the regurgitation cases. Creamy-white specks in the pharynx of two young pigeons were thought to have been due to avitaminoses A associated with a too sudden change-over from maternal feeding to the predominantly whole maize or pea diet upon which the adults were fed, but possibility of chronic pasteurellosis, infectious laryngotracheitis, and aspergillosis could not be entirely discounted.

One case of foreign body (a fine gold chain) in a parrot, lodged distal to the proventriculus, caused an enteritis and *post-mortem* examination revealed a coincident or resultant avitaminosis A. A recently imported young parrot provoked a psittacosis scare by its symptoms, but *post-mortem* examination showed only one small necrotic focus in the bird's liver to account for the marked digestive and respiratory symptoms with great depression it had exhibited. No significant bacteria were isolated and mouse inoculation was negative. A third parrot, 40 years old and showing little signs of ill-health save lethargy, a disinclination to preen, and an offensive mouth odour, was found to have a mass of impacted food material and epithelial cells jammed tightly under the tongue and against the lower beak. No fungal hyphae could be found and it was thought that a crumbly wet diet was the cause of impaction. A 22-year-old Amazon parrot on an abnormal diet showed recurrent prolapse of the cloaca due to prolonged diarrhoea and tenesmus.

Distension of the abdomen in two budgerigars resulted in breakdown of the abdominal musculature. In one was a herniated loop of bowel distended with soft faeces which had to be emptied daily by manipulation. Autopsy of this case showed a chronic peritonitis from which a haemolytic staphylococcus was isolated, and an enlarged and ruptured liver. In two budgerigars presented in dying condition, enlarged liver showing congestion and necrosis was found, and from the heart blood pneumococcus was cultured.

With crop stagnation and distension regurgitations, digital pressure was usually sufficient to empty the organ. Aspiration of fluid with a wide-bore hypodermic needle was carried out in one case and a weak solution of potassium permanganate injected to prevent further fermentation. With dry impactions, the crop was incised, emptied, and resutured. Where brown fluid regurgitation persisted, sulphadimidine 5 per cent. with kaolin and creta praeparata 3 drops 3 times a day was found to be effective. Coprophagia was treated by a mixed vitamin addition to the diet. All the above birds were put on a more easily digested diet, powdered dried yeast (heat killed), a little fruit and green food, milk, a little soaked seed, and occasionally aneurin hydrochloride to promote tone in the crop wall in cases of prolonged dilatation.

With suspected cases of nutritional roup, cod-liver oil was used as a source of vitamin A, and a varied and gradual change-over to adult food was advised. Soaking or boiling the grain and peas was also favoured for the first week after leaving the nest.

Respiratory Disorders

Neoplastic or other mechanical conditions causing respiratory distress mainly by pressure and cachectic and septicaemic dyspnoeas are not included under this head. Four budgerigars, one parrot, and a linnet showed on *post-mortem* examination pneumonia and necrotic foci of the lungs and/or consolidation of air sacs with cheesy material and an abundant fungal mycelium was found in the lesions; an Aspergillus species. Five of the affected birds were less than one year old. In the remaining respiratory case no pathological examination was available. Rapid, shallow, or heaving respiration, beak agape, huddling with ruffled feathers, with or without a slight mucoid diarrhoea was the typical clinical picture, but identical symptoms accompanied neoplasia involving the thorax and so careful palpation was necessary for differentiation.

It seems that true lung, or air sac, infection has a high mortality, little if any influenced by available drugs, and fungal or other chronic infections form a large part of respiratory troubles. It is perhaps worth speculating on the source of the fungal and other chronic air-sac and lung infections. In young birds, they are probably infections acquired in the breeding establishment where the infection is endemic and that this infection is ignited by the sudden change of environment. In older birds, seed contamination may be the source, at least with fungal infections.

An important clinical point is that progressively more laboured respiration over at least a week, even if intermittent improvement occurs, usually means one of two things—abdominal neoplasia or lung and air-sac infection. With either of these prognosis is grave, and mere handling of the bird is a hazard.

Nervous Abnormalities

This group comprised of conditions affecting mainly the power of perching, walking, flying, and postural maintenance, for which there was no evidence that trauma was responsible. The title "nervous" is used tentatively for in no case was there proof of definite lesions of the nervous system. All but 9 were under 2 years of age. Of this 9, in 4 birds (2 budgerigars of 5 and 7 years of age, respectively, and two canaries aged 8 years) it was thought that senile changes might have accounted for the symptoms which included an inability to fly and poor perching power in legs; no grasp in claws for perching; extensor weakness of the limbs preventing standing or perching.

Nine young budgerigars had weak or absent gripping reflexes in one or both legs, varying from slight weakness to an almost complete absence of normal perching reflexes. In these complete flaccidity simulated rupture of the tendo-achilles. Five birds, each a few months old, showed curling of the digits into a clenched position which prevented perching and made walking barely possible on the hocks and clenched claws. In two of these, one foot curled several days before the other and it was a gradual process. A dietary deficiency, largely of B_1 and other B-group vitamins was suspected. One bird perched with its body horizontal, and when it flew it landed on its beak. A cerebellar lesion, possibly acquired by flying into a wall, was suspected. Four birds were suspected of having defective vision for barely perceptible pupillary reaction to strong light was noted. An interesting case was that of a green African parrot which showed unmistakable chorea of the left wing. No other symptoms were seen. Another showed epileptiform convulsions of increasing frequency after 3 weeks of diarrhoea which resulted from an abnormal diet consisting mainly of fruit.

Treatment was on similar lines in almost all cases. Yeast, aneurin hydrochloride, egg, and other animal protein supplements were advised on the diet sheets and, in the optically affected birds, vitamin A in the form of cod-liver oil. Relative rest in a smooth-walled box with a perch on the floor was advised to encourage rehabilitation. The parrot with epileptiform convulsions had no further fits beyond 36 hours of commencing feeding a mixed vitamin supplement. Several were returned for a check on their progress and improvement was generally seen within a fortnight, in some cases complete normality being regained.

Reproductive Disorders

As the birds dealt with were practically all pets kept in isolation it is not surprising that the number of reproductive disorders was small.

Two of the 6 birds of this group were egg-bound and in the other 4 there was cystic dilatation of the oviduct which appeared as large, fluctuating swellings ventral to the cloaca. In 2 of these, marked dyspnoea was also present.

In the egg-bound cases the application of oil and manipulation failed to release the egg. In one case the egg was deliberately carefully broken and the shell removed piecemeal; in the other, the oviduct was incised over the everted part and the egg removed whole. Neither was brought in for further treatment. In the other cases an incision was made over the distension (two under light ether anaesthesia, and the others with respiratory distress, without anaesthesia). The dilatations were ligated with fine catgut, and the sac-like structures removed. The abdominal wound was then closed in two layers, fine continuous catgut for the muscles and fine, interrupted gut sutures in the skin. Healing was almost complete in all 4 cases within about 6 days. In one of these birds, the sacculation was multi-locular containing a clear watery fluid and white granules, and the others fluid and inspissated yolk. Two of the birds showed some evidence of regrowth two weeks later, and in one as growth continued the bird was destroyed. The other birds have been seen alive and well several weeks after operation. Histological examination showed columnar epithelium with a fimbrated edge of the oviduct type.

Three cases of sterile peritonitis included in the miscellaneous group were thought to have been caused by free egg material.

Developmental Abnormalities

Six budgerigars and one sparrow were seen with anatomical deformity of the limbs. In view of the youth of the birds—the oldest was 2 months of age—they were tentatively classed as congenital.

They comprised:—

(a) Spreadeagling of the legs with deformity of the sternum, ribs and pelvis, and/or head of femur such that the body was dorso-ventrally compressed.

(b) Lateral projection of the femur with normal stifle movement. On being placed in the normal parallel horizontal position, the femurs on release sprang back to the original abducted position, 3 cases.

(c) Intermittent luxation of the tarsal joint, 2 cases.

(d) Deformity of the right stifle and upper third of the tibio-tarsus with outward rotation of the lower part of the limb.

The significance of this small group is not great but a number of hereditary conditions may well be perpetuated by the sale of and breeding from affected or carrier birds.

Unclassifiable

This heterogeneous collection includes vague functional, alimentary, neoplastic, traumatic, reproductive, and other troubles and malaise of too ephemeral an existence to be classified, and at times even beyond description. No useful purpose would be served by analysing them.

Notes on the General Handling and Sexing of Cage-birds

Although the handling of small cage-birds is generally approached with trepidation because of the widely accepted belief that they will die therefrom, in this series only one bird, a budgerigar, died as the direct result of handling for examination. The lethal factor in handling small birds appears to be

pressure on the thorax while attempting to prevent escape. The tendency to counter the bird's efforts to escape is to squeeze the fingers tighter and tighter. The bird's reaction is to panic with the increasing effort of every breath and anoxia results. The lungs become congested and the bird dies of cardiac failure secondary to anoxia, venous congestion, and shock.

Even the smaller varieties of psittacines can inflict a very painful peck. For parrots, unless very well-tempered, gloves are indicated for approaching the bird in the cage. In this species and the budgerigars, it is advisable to approach from behind the head, back of the hand facing the same direction as the bird. The last few inches are best accomplished briskly and the index and second fingers are now held firmly astride the neck. The thumb and third and fourth fingers can be arranged under the thorax and abdomen as a loose but rigid cage. A wing or leg can now be held between any two digits as required for examination purposes. Any attempt to peck is countered by extending the first two fingers, and thereby, the bird's neck. In this way the head, legs, wings, and ventral aspect can readily be inspected as also can other parts by slight rearrangements of thumb and third and fourth fingers. Often, simple surgery can be performed under this restraint. With species with less powerful beaks—canaries, finches, and the like—this method is equally applicable with safety of the bird. A fair degree of unhampered twisting and fluttering can be permitted within this rigid cage-work of fingers, but is easily controlled during operation.

Sudden death within a few minutes of injection of 40,000 units of streptomycin in 3 budgerigars, and 100,000 units in a parrot, all in the pectoral muscle, led the writer to fear that the shock of injection was the cause of death. All 4 birds, and another which died after oral administration, were systemically very ill. Later on, when successful results were obtained in treatment of extensive accidental and surgical wounds, even intra-abdominal ones, where wound dressings of penicillin G and sulphadimidine were used, it was decided that the fatalities were more likely due to a streptomycin toxicity, accentuated by the weak condition of the birds. As 3 of the birds showed extensive lung and/or air-sac affections with cardiovascular sequelae on *post-mortem* examination, it might well be concluded that the restraint and fear it provoked could have accounted for the shock and circulatory collapse. Nevertheless, in view of these fatalities, injection of antibiotics has subsequently been little used and oral and topical applications of penicillin and sulphonamides are preferred. The use of streptomycin in any form has been suspended for the present. Oxytetracycline has been found helpful in some alimentary disturbances, administered as a suspension of a "terramycin animal food supplement." Other antibiotics have not been sufficiently tested to comment fairly on their values.

It is impossible to sex budgerigars in the first 2 months of life (*i.e.* for a few weeks after leaving the nest at which age they are often sold). At about 2 months the dull blue cere of both sexes changes: in the male it becomes a brighter blue and in the female it fades, becomes whitish, then a light tan, and finally a brown colour.

After the first month, the youngsters become more and more like their parents. The tiny black stripes which are present on the forehead in the young commence to disappear at 6 weeks of age becoming bright yellow in greens and yellows, or white in blue birds.

The cere in females out of the breeding season may become pale blue. Additionally, as experience with this series shows, in an ailing male the cere often becomes a reddish brown with varying degrees of laminated deposit, described elsewhere as brown hypertrophy of the cere. This deposit can be removed, revealing a pale blue cere underneath. This accounts for some of the "sex unknown" figure for this species.

The sexing of canaries may be difficult for beginners and especially at certain times. At breeding time the male cloaca is pronounced and protrudes downwards. In the female, the vent is swollen and rounded almost to the level of the abdomen. In the young the difference is scarcely discernible. The surest way is to hear the male sing. Females occasionally sing and are said to make bad breeders if they do Sometimes at about 5 weeks in males, the throat may swell up as if in an effort to sing, but this often does not occur until after the first moult.

Acknowledgments.—My grateful thanks are due to Professor J G. Wright for the continued encouragement and editorial advice he has given me in this work; to the staff of the department of Veterinary Pathology under Professor D. L. Hughes and particularly Dr. G. O. Davies, Mr. H. P. Harding, Mr. J. M. K. Mackay, and Mr. G. Weston for their lively interest and examination of pathological material and advice regarding it; to Mr. R. B. Griffiths and Mr. M. J. Lavoipierre for their help with parasitological problems; and finally to Mr. A. C. Shuttleworth for his kindness and encouragement in allowing me to deal with all the bird cases brought to the Clinic for treatment.

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Current Literature

BOOK REVIEWS

Methods of Typing and Cultivation of Foot-and Mouth Disease Viruses. Published by the European Productivity Agency of the Organisation for European Economic Co-operation (O.E.E.C.), Paris, 1957. 7s.

A seminar, organised by the European Productivity Agency of O.E.E.C. was held at the Netherlands State Veterinary Research Institute, Amsterdam, in November, 1955. Its title was "Methods of typing and cultivation of foot-and-mouth disease viruses," since (as the preface states) these subjects are of fundamental importance in controlling this disease. The report recently published records the technical papers read at the seminar and a summary of the discussions and conclusions reached by the participants.

Part I of the report deals with general considerations, summary of discussions, and the conclusions. After stressing the necessity of international collaboration in a system of warning, exchange of strains, and vaccine production, this part of the report goes on to deal with standardisation of nomenclature, identification methods, and methods of virus culture. As regards methods of identification the basic procedure is the complement fixation test, which may be performed on the original epithelium or on material obtained after the passage of the original through mice, tissue culture, or cattle. The method finally used depends on the speed of identification required and on economic considerations. Various procedures of the complement fixation test are employed, and the delegates thought it more important that standard reagents should be used rather than a standard procedure, and that the preparation of reference antisera and maintenance of strains should be entrusted to one laboratory, which, it was unanimously agreed, should be the Research Institute (Animal Virus Diseases), Pirbright.

The importance of serum neutralisation and cross immunity tests was also stressed in the identification of new types and in the establishment of variants, and criteria were laid down, which new types and variants must satisfy. In the study of culture methods of foot-and-mouth disease viruses all methods were discussed. These methods include production of virus on living animals such as on the tongue or skin of cattle, in calves or in small laboratory animals, and cultivation of the virus *in vitro* by tissue culture methods. Some methods were suitable for large-scale production, some more suited for titration and identification procedures, and some were still at the research stage. The conclusions to this part represent the essential points of the discussion and those of immediate application. This first part of the report gives a concise, useful but uncritical summary of the methods of typing and cultivation of virus.

Part II consists of the technical papers presented at the seminar. Among those dealing with the complement fixation test is a paper by Brooksby and Rogers, in which they report the identification of a new type of virus (the seventh)—Asia I. There is an interesting paper on the serum neutralisation test in tissue culture by van Bekkum, and papers on *in vitro* and *in vivo* methods of virus cultivation by Dutch, Belgian, French, and German workers. One debatable point emerging from work on the Frenkel method of *in vitro* virus cultivation is that some workers claim that vaccines containing virus recently passaged in animals are more immunogenic than vaccines containing virus that has had many passages in tissue culture. Some papers tend to be partisan, which detracts from their scientific value, and as a whole the papers should be regarded more as a basis for discussion than as a presentation of detailed results.

Richtlinien fur die Sektion der Haustiere. (Guide to the *post-mortem* examination of domestic animals). J. DOBBSTEIN. 8th revised edition. Pp. 129 with 39 text figures and 4 tables. D.M. 13.80. Berlin and Hamburg: Paul Parey.

This is the 8th edition, incorporating some new material, of a very handy guide to the *post-mortem* examination of domestic animals. The examination of the horse at autopsy is described in most detail, but the other domestic animals, including rabbits and poultry, are also well covered. In general, after a brief résumé of the anatomical situation, the method of removing the various organs is described. The normal features are very clearly indicated by a sort of catechism, a series of questions being posed to which a positive answer is usually possible if the organ concerned is quite normal. Variations from the normal, and their significance, are briefly but clearly described.

There are additional chapters in which are dealt with such matters as the method of sending in material for laboratory examination. Tables and diagrams are used to indicate the appearance, weight, and size of important organs, and the ageing of animals by the teeth is described.

This is not claimed to be an exhaustive treatise, but it is an extremely useful and convenient book, particularly for the German-reading veterinary student during his pathology course.

Fundamentals of Immunology. By W. C. BOYD. Third edition. Interscience Publishers Ltd., London. 75s.

Since the publication of the 1st edition in 1943, Boyd's Immunology has become one of the 3 or 4 standard works on the subject in the English language. Though not intended as a comprehensive review, it can be relied on to give a clear picture of our present knowledge in any one of the many branches of this complex science. For the research worker who studies immunology or uses it as a tool the new, enlarged edition is almost indispensable; current trends and theories are expertly surveyed, and the copious bibliography includes most of the key references.

Professor Boyd doubtless knows the capacities of his own students, but many teachers at the medical and veterinary schools will feel that the present edition is far too complicated for their classes. Moreover, it sometimes tends to stress recent research

into relatively trivial matters at the expense of the real fundamentals. The section on toxins is a case in point. There are several pages summarising research on the chemical and physical properties of toxins, but a student could easily miss the essential point about a toxin, which is that it must be toxic! The author reaches his fifth paragraph before casually introducing this vital concept as a subsidiary clause in a sentence about something else.

Numerous examples are drawn from human medicine but animal diseases are scarcely mentioned. A pity, this, for the chapter on practical uses of immunity could have been richer for a few examples from the veterinary field, in which vaccination is probably more widely practised than in human medicine. In the section on diagnostic methods an account of the toxin neutralisation tests used in the diagnosis of enterotoxaemia in sheep would surely have been worth while, for the procedure has no parallel in human medicine except in the diagnosis of botulism (also unaccountably omitted). Again, it is odd that an author renowned for his work on human blood groups should have nothing to say about a tempting question like that of haemolytic disease in foals.

The system of headings is clear and logical, and makes a difficult subject easier to follow. The present edition contains several new chapters, including one on autoimmunisation, and the last hundred or so pages comprise a sort of immunological cookery book of instructions for various techniques; surprising, perhaps, in a book on fundamentals, but interesting. There are plenty of tables, mostly excellent, and the illustrations are few but good, except for a rather pointless photograph of "models" of antibody molecules, and 2 maps, atrociously printed as half-tone plates.

Review: Proceedings of Conference: "Agriculture in British Economy." Publishers: I.C.I. Limited, 2, Buckingham Gate, London, S.W.1. 21s.

The proceedings of the above Conference are now issued in book form, and cover a very wide field of interests. Titles of papers include "Agriculture's Place in the National Economy," "The Sources of our Food Supplies," "The Use of Manpower," "Capital Needs of the Industry," "Government Support," "Price Policy and General Financial Matters," and a series on the future of arable crops, pigs, poultry, milk, beef, and sheep. The papers, given by authorities of the day, were extensive and comprehensive. Views, in brief, of some of the authors give an idea of prevailing trends.

It was felt that farmers should cease growing a little of everything and specialise in products in which they are best suited, but the benefits of mixed husbandry should not be sacrificed by going too far in this direction. World food supplies seem to be more likely to improve than to deteriorate, or the conditions of trade more likely to get better than worse, from our point of view as an importing country. In fact there is a genuine danger of this expansion resulting in overproduction and the consequent emergence of surplus stocks of certain commodities. The demand for more attractive and nutritious foods has been increased from all social classes, but that for cheaper and more starchy foods, such as cereals, potatoes, etc., has received a check.

It was felt that research programmes should not be cut down, but should in fact be intensified, and that greater efforts should be made to disseminate knowledge already available and to see that this is applied.

Referring to the future of the pig industry, figures are given indicating that 2 litters per sow per year in Denmark are the average, as compared with 1.8 in Britain, and of 15.5 pigs reared to slaughter per sow per year in Denmark, 12.2 reach this stage in Britain. There is no reason why pigs should not be weaned at 6 weeks. Fertility in swine, according to the British Veterinary Association's booklet, is declining, and pre-natal death losses may amount to one third of the young starting as fertile eggs.

During the next 10 years, the poultry industry should consolidate rather than expand, as production of eggs and poultry has equalled requirements. Until markets can be expanded or costs reduced, it is desirable to avoid stimulating further the production of eggs. Indifferent hatchability is a serious international problem, 1 in 3 set for incubation failing to hatch; 10 per cent. improvement in this respect would save the industry £1,000,000. One speaker thought that the poultry industry should expand, but should be put on a strictly business-like basis so far as production and marketing were concerned.

Turning to dairying activities, it was felt that cheaper production of milk by more and better use of grass should be achieved. This is easier in areas where the rainfall is high. In remote areas production should be directed towards summer milk, for it is cheaper and essential for dairy produce. Improvement in grassland management offers a wonderful opportunity to milk producers for summer milk. At the moment, production from grassland in the U.S. is low at 15 cwt. of Starch Equivalent per acre. This could be raised by at least one third. It was thought that there should be a shift from grass conservation in terms of hay to silage—not solely on a nutritional basis, but because hay-making is incompatible with good grassland management, whereas cutting grass for silage is an indispensable technique. The emphasis should be on the quality of grass rather than on more grass.

The national and individual significance of increasing net output of livestock from farmers must have much wider appreciation, or we should be forced by circumstances beyond our control to return to the practice of feeding our stock through our land. Ten per cent. increase in our present tillage acreage should be attempted over the next 10 years. Such additional production should be diverted to livestock, so cutting down imported feeding-stuffs. On the other hand, a feature which is often overlooked is that the importation of feeding-stuffs into the U.K. means the importation of fertility through manurial residual values of such foods. An increase in home-grown feeding-stuffs is quite possible but this should not replace cash crops sold off the farm, such as sugar beet, potatoes, etc. The increase should be through the ploughing up of worn out or impoverished grassland.

News and Comment

THE HARRY STEELE-BODGER MEMORIAL PRIZE

This prize was created by the fund established in 1953 to perpetuate the memory of the late Harry Steele-Bodger. It takes the form of a travelling scholarship or a contribution to a tour abroad. In addition, applications for grants of small amounts to enable practising members of the profession to make short study tours abroad receive sympathetic consideration. Members of the Royal College of Veterinary Surgeons are eligible, together with final-year students of any of the University Veterinary Schools in the United Kingdom and Eire.

Applications, giving an estimated cost of the proposed study tour, should be made to the General Secretary, British Veterinary Association, not later than July 31st 1958.

THE ROYAL SOCIETY OF HEALTH

A Sessional Meeting will be held on Monday, March 10th, at 2.30 p.m., in Caxton Hall, Caxton Street, Westminster, S.W.1, when the following symposium will be presented and discussed: "Nuclear Radiation Hazards: Training of Local Authority and Hospital Personnel."

Papers will be read by Sir Ernest Rock Carling, F.R.C.S., F.R.C.P., Chairman, World Health Organisation Committee on Post-graduate Training in the Public Health Aspects of Atomic Energy, and former Chairman of the International Commission on Radiological Protection; Mr. G. B. Courtier, B.SC., D.I.C., F.R.I.C., M.R.S.H., Senior Principal Scientific Officer, Public Health Department, London County Council; and Dr. W. H. P. Minto, D.P.H., M.R.S.H., County Medical Officer, Cumberland County Council.

The Chairman will be Dr. J. A. Scott, O.B.E., Q.H.P., F.R.S.H. (Chairman of the Society's Council).

NAPT COMMONWEALTH CHEST CONFERENCE

The fifth Commonwealth Conference of the National Association for the Prevention of Tuberculosis will be held at the Royal Festival Hall, London, from July 1st to 4th, 1958.

More than 2,000 delegates are expected, and the Conference will offer unique opportunities for those interested in tuberculosis, diseases of the chest and heart, and problems of preventive medicine, to meet workers in similar fields throughout the world.

The Conference fee for full membership is £5 5s. or 15s. per session, *if the registration fee is received by May 31st, 1958*. The late registration fee will be £6 6s. or 17s. 6d. per session. Delegates will be asked to state on the registration form whether they wish to order a copy of the transactions of the Conference at an additional charge of 15s. The volume will be published only if sufficient orders are received.

IN PARLIAMENT

Meat (Inspection)

SIR F. MEDLICOTT (January 27th) asked the Minister of Agriculture (1) what proportion of the total amount of meat passing through slaughterhouses is actually inspected; and (2) what proportion of meat inspected at slaughterhouses is found diseased and unfit for human consumption.

THE JOINT PARLIAMENTARY SECRETARY TO THE MINISTRY OF AGRICULTURE, FISHERIES AND FOOD (MR. J. B. GODBER): It is estimated that the proportion of meat inspected is about four-fifths of the total, of which less than one per cent. is found unfit for human consumption.

SIR F. MEDLICOTT: Does my hon. Friend not feel that the position is rather unsatisfactory? Does it not follow from these figures that a certain amount of diseased meat must inevitably find its way into the shops, and can he not advance the date when all meat going into slaughterhouses is inspected?

MR. GODBER: I entirely agree that we must try to get all meat inspected at the earliest possible date. We are doing all we can to that end, and at the moment we are having consultations with those concerned to see whether we can speed up this matter.

Surplus Milk (Meat Production)

MR. DE FREITAS (January 27th) asked the Minister of Agriculture, Fisheries and Food if, in view of the importance of using surplus milk for the production of milk-fed veal, he will inquire into the possibility of a scheme by which farmers may receive payment for meat in monthly instalments on the lines of the present practice of the Milk Marketing Board in respect of milk.

MR. GODBER: My right hon. Friend recognises the importance of using surplus milk for veal production and for rearing calves for beef. So far as concerns schemes for advance payments on fat cattle, two small pilot co-operative ones are being launched under the auspices of the Agricultural Central Co-operative Association, and I believe that larger-scale arrangements operated on behalf of farmers are under consideration by a national organisation.

Crystal Violet Vaccine

SIR A. BOSSOM (January 31st) asked the Minister of Agriculture, Fisheries and Food if he will permit supplies of crystal violet vaccine to be made available to farmers to combat swine fever.

MR. JOHN HARE: Crystal violet vaccine is suitable for use only in skilled hands and for this reason the supplies produced at my Department's veterinary laboratories are sold only to veterinary surgeons. I do not control the distribution of the small quantities of crystal violet vaccine imported under licence.

Foot-and-Mouth Disease

MR. PEYTON (February 3rd) asked the Minister of Agriculture, Fisheries and Food to what causes he attributes the recent outbreak of foot-and-mouth disease in south-east Somerset.

THE JOINT PARLIAMENTARY SECRETARY TO THE MINISTRY OF AGRICULTURE, FISHERIES AND FOOD (MR. J. B. GODBER): This may have been either a primary outbreak due to infection from the Continent or, alternatively, may have been a secondary outbreak caused by the series in Dorset.

MR. PEYTON: Does not my hon. Friend agree that this has been an exceedingly serious outbreak, and does he not appreciate that the very heavy losses involved have given rise to considerable anxiety, particularly about whether the outbreak has been caused by germs from imports of infected Argentine meat? Will he go into the matter, since I can assure him that there is considerable concern about it, more so than I have ever known?

MR. GODBER: Yes, I recognise that this outbreak has caused very grave concern and that there are losses quite outside those which we can compensate. We are considering this very seriously, but the only evidence we have on this outbreak points to infection through birds from the Continent, which is something, of course, we cannot control.

Meat (Inspection)

MR. HAYMAN (February 3rd) asked the Minister of Agriculture, Fisheries and Food what are the percentages of uninspected meat passing through slaughterhouses in county boroughs and outside county boroughs, respectively.

MR. GODBER: The information is not readily available, but I will write to the hon. Member as soon as it has been obtained.

MR. CHAMPION: Is not the Joint Parliamentary Secretary aware that it is disgusting that uninspected meat should reach the public? Will he not take advantage of the Slaughterhouses Bill now going through the House to ensure that no uninspected meat reaches the public?

MR. GODBER: I agree with the hon. Member that we want 100 per cent. meat inspection. We are as keen to see that as is anyone else. However, there are difficulties, and I do not know that the Slaughterhouses Bill can absolutely ensure it. We are having a good deal of advice on that matter from some of the hon. Member's hon. Friends, and I am sorry that he is not with us in the Committee.

MR. HAYMAN asked the Minister of Agriculture, Fisheries and Food what is the estimated quantity of uninspected meat passing through slaughterhouses and becoming available for human consumption.

MR. GODBER: I cannot give a precise figure, but we estimate it is about one-fifth of the total meat passing through slaughterhouses.

MR. WILLEY: While appreciating the steps taken by the Department, will the Joint Parliamentary Secretary give favourable consideration to the new Clause to the Slaughterhouses Bill which we intend to put down to provide for complete inspection?

MR. GODBER: I am afraid that I cannot be drawn as far as that.

Condemned Meat (Marking)

MR. J. E. B. HILL (February 3rd) asked the Minister of Agriculture, Fisheries and Food whether he will seek powers to re-introduce the practice of staining and of regulating the disposal of meat that has been condemned as unfit for human consumption.

MR. GODBER: My right hon. Friend has this matter under active consideration in consultation with his right hon. and learned Friend the Minister of Health.

MR. HILL: Can my hon. Friend tell me when he hopes to reach a decision in this matter? Is he aware that if he decides in favour of restoring the regulation, it will give satisfaction not only to the public at large but also to the trade associations concerned, since they are very anxious to minimise unfair trading and bad practices? Does he not also agree that although it is a very important problem it is not large, since the amount of meat unfit for human consumption is only a minute fraction of total supplies?

MR. WILLEY: While appreciating the importance of a speedy decision, will the Joint Parliamentary Secretary, nevertheless, review the general regulations affecting diseased meat to see whether we can follow the course taken up by many European countries?

KENNEL CLUB RULING ON ENTIRE DOGS

An announcement recently made by the Secretary of the Kennel Club is of considerable importance to the profession, the more especially as it represents the successful culmination of conversations between the Kennel Club and the B.V.A. The announcement reads as follows:—

"I am directed by the committee of the Kennel Club to announce that the existing directive to judges instructing them to ignore the condition of monorchidism/cryptorchidism, which has been in force since 1924, shall be withdrawn forthwith and therefore judges who have knowledge of this condition may penalise dogs for it if they wish, although at the moment it is not grounds for disqualification.

"I am further instructed to add that the committee is now deciding the details of further legislation which will mean that as from January 1st, 1959, any male dog which is exhibited at a show and which is not entire (*i.e.* an entire dog is one which has both testicles descended and in the scrotum) shall be disqualified.

"Show regulations are being amended for championship, licence, and sanction shows and these amendments will come into force on January 1st, 1959."

The profession has, for some years, been disturbed by the increase in monorchidism and cryptorchidism amongst pedigree dogs, and last year the B.V.A. brought its views to the notice of the Kennel Club. The Club invited our Association to submit evidence and to express an opinion on the hereditary nature of the condition. Having received our views the Club decided at first to approach the subject with caution but decided as a preliminary measure that any pedigree dog for export must have a certificate stating

that the dog was entire. At this time the American Kennel Club had imposed a rule prohibiting the exhibition of monorchid and cryptorchid dogs in United States shows.

There followed further discussions between representatives of the B.V.A. and the Kennel Club which resulted recently in the announcement made above.

It may be noted in passing that the terms monorchidism and cryptorchidism tend to be misleading, whereas the new definition "entire" is self-explanatory.

When, in 1959, the new rule comes into force, it will not, of course, eliminate the condition, but should have the effect of gradually reducing it. It will bring the practice in this country into line with that of a number of overseas countries, and will, incidentally, make the conditions for pedigree dogs similar to those for other pedigree animals.

VETERINARY WIRELESS TRANSMITTER ON PENRITH BEACON

Two Penrith veterinary surgeons have obtained permission to erect a mast for a V.H.F. wireless transmitter on Penrith Beacon. The apparatus will make it quick and easy to transmit messages so that in emergency, help can be directed to distant farms. The county secretary of the National Farmers' Union is reported as having expressed great satisfaction at the arrangement, as have a number of individual farmers.

Many other veterinary surgeons have, of course, transmitting and receiving sets fitted in their cars.

THE REGISTER OF VETERINARY SURGEONS

Under the provisions of Section 25 (4) of the Veterinary Surgeons Act, 1948, and Statutory Instrument No. 1,729 of 1957, the name of the undermentioned was restored to the Register of Veterinary Surgeons on January 31st, 1958:—

Mr. Wilfred Gordon Willoughby Aston Woollett, c/o St. Mary's Place, Church Walk, Ulverston, Lancs.

MRS. G. A. RAILTON

The funeral took place at St. Peter's Church, Glasbury-on-Wye, on January 6th, of Mrs. Georgina Alice Railton, who died after a long illness. A native of Newtown, Montgomery, she was the wife of Mr. J. P. Railton, M.R.C.V.S., who qualified in 1910, and who practised in Builth Wells and Kington before going to Glasbury in 1944. He, with a son and daughter, survive her.

In her younger days Mrs. Railton was an accomplished hostess, and leading members of the profession and other persons of note enjoyed the hospitality of her husband and herself. Through many contacts, and through her personal graciousness she did a great deal in an unassuming way to maintain the prestige of the profession wherever her husband practised.

PERSONAL

Mr. E. Cotchin, College Reader in Veterinary Pathology at the Royal Veterinary College, who is honorary treasurer of the B.V.A., has been awarded the doctorate of science in the University of London, for his work in pathology with particular reference to neoplasia in domestic animals.

Births

HOGG.—On January 16th, 1958, to Kathleen, wife of Alex. H. Hogg, M.R.C.V.S., Leamington Spa, a daughter, Caroline Mary, sister for David.

PRYCE.—On January 27th, 1958, to Patsy, wife of Ray Pryce, B.V.SC., M.R.C.V.S., of Bath, a son, brother for Simon.

SEDGWICK.—On January 27th, 1958, at Helme Chase, Kendal, to Anne Jennifer, R.G.N., wife of John R. W. Sedgwick, B.SC., M.R.C.V.S., Queen's Drive, Windermere, a son, John Richard William.

Marriage

BOARDMAN—THORNTON.—On Saturday, January 25th, 1958, at Four Oaks, Warwickshire, Anthony Charles, B.V.M.S. (GLAS.), M.R.C.V.S., son of Mr. and Mrs. L. C. Boardman, of Alvechurch, Worcs., to Diana, M.R.C.S., L.R.C.P., eldest daughter of Dr. and Mrs. Pensam Thornton, of Sutton Coldfield, Warwickshire.

COMING EVENTS

February

12th (Wed.) Meeting of the West of Scotland Division at the University School of Veterinary Science, Garscube, Bearsden, Glasgow, 2.30 p.m.

Meeting of the R.A.V.C. Division at the Central Veterinary Laboratory, New Haw, Weybridge, Surrey, 2.15 p.m.

13th (Thurs.). Spring Meeting of the Dumfries and Galloway Division in the Crown and Mitre Hotel, Carlisle, 2.15 p.m.

14th (Fri.). General Meeting of the Society for the Study of Animal Breeding at the B.V.A., 7, Mansfield Street, Portland Place, London, W.1, 11 a.m.

Joint Meeting of the Society of Practising Veterinary Surgeons and the Shropshire Veterinary Club at the Radbrook Hall Hotel, Shrewsbury, 7.30 p.m.

General Meeting of the Society of Practising Veterinary Surgeons in the Radbrook Hall Hotel, Shrewsbury, 8 p.m.

18th (Tues.). 89th Annual General Meeting of the Eastern Counties Veterinary Society at the Bell Hotel, Norwich, 10 a.m.

19th (Wed.). Meeting of the Section of Comparative Medicine, Royal Society of Medicine, at the Society's House, 5 p.m.

Meeting of the Warwickshire Veterinary Club at the Regent Hotel, Leamington Spa, 8 p.m.

20th (Thurs.). Ordinary Meeting of the Ayrshire Veterinary Association in the Ailsa Lounge, Ayr Station Hotel, 2.30 p.m.

Annual General Meeting of the South Eastern Veterinary Association at the Royal Star Hotel, Maidstone, 2.30 p.m.

General Meeting of the Herts and Beds Veterinary Association at 70, Holywell Hill, St. Albans, 7.30 p.m.

21st (Fri.). Annual Ball of the Liverpool University Veterinary Society in the Students' Union, 8 p.m.

26th (Wed.) Ordinary General Meeting of the Southern Counties Veterinary Society at the Polygon Hotel, Southampton, 7 p.m.

Meeting of the A.V.T. & R.W.'s Scottish Regional Group in the Moredun Institute, Gilmerton, Edinburgh, 9, 2 p.m.

27th (Thurs.). Meeting of the Essex Veterinary Society at the Essex Institute of Agriculture, Writtle, Essex, 7.30 p.m.

28th (Fri.). Dinner-Dance of the South Eastern Veterinary Association in the Queen's Hall, Royal Star Hotel, Maidstone, 8 p.m.

Annual General Meeting of the Royal Counties Veterinary Association at the Grosvenor Hotel, Kidmore Road, Caversham, Reading, 2.15 p.m.

THE COLONIAL VETERINARY SERVICE

Promotions. C. L. Bent, Veterinary Officer, Jamaica, becomes Senior Veterinary Officer, Jamaica; A. C. Lewis, Veterinary Officer, Jamaica, becomes Senior Veterinary Officer, Jamaica; L. T. McDonnough, D.V.M., D.T.V.M., Senior Veterinary Officer, becomes Director of Veterinary Division, Jamaica.

Transfers. J. E. R. Roe, O.B.E., Director of Veterinary Services, Bechuanaland Protectorate, becomes Director of Veterinary Services, Tanganyika; J. G. Black, D.V.M. (TORONTO), D.V.P.H. (TOR.), D.V.S.M., Veterinary Officer, Northern Rhodesia, becomes Livestock Improvement Officer, Kenya.

New appointments. M. R. Dexter, Veterinary Officer, Nyasaland; I. G. Gibson, Veterinary Officer, Kenya; E. P. S. Rogers, Veterinary Officer, Tanganyika.

FOOT-AND-MOUTH DISEASE

The areas described in the schedule below are declared to be Infected Areas for the purpose of preventing the spread of foot-and-mouth disease:—

IN THE COUNTY OF SOMERSET

The City and County Borough of Bath.

The Borough of Glastonbury and the City and Borough of Wells.

In the Petty Sessional Division of Axbridge: The parishes of Wedmore, Cheddar, Burrington and Blagdon.

In the Petty Sessional Division of Wells City and County: The parishes of Walton, Street, Sharpham, North Wootton, Meare, Godney, Wookey, Rodney Stoke, Westbury, St. Cuthbert Out, Dinder, Priddy and Chewton Mendip.

In the Petty Sessional Division of Shepton Mallet: The parishes of Emborough and Binegar.

In the Petty Sessional Division of Temple Cloud: The parishes of Nempnett Thrubwell, Ubley, Compton Martin, West Harptree, East Harptree, Litton, Chew Stoke, Stowey, Hinton Blewett, Ston Easton, Cameley, Clutton, High Littleton, Timsbury, Farrington Gurney, Paulton and so much of the parish of Chew Magna as lies to the south of the main road running from Winford *via* Chew Magna to Whitchurch, including the said road.

In the Petty Sessional Division of Kilmersdon: The parish of Chilcompton and so much of the parishes of Stratton on the Fosse and Norton-Radstock as lies to the north and west of the main road running from Shepton Mallet *via* Radstock to Bath, excluding the said road.

In the Petty Sessional Division of Bridgwater: The parishes of Shapwick, Ashcott and Greinton.

In the Petty Sessional Division of Shepton Mallet: The parishes of Ashwick, Croscombe and Pilton and so much of the parish of Shepton Mallet as lies to the west of the road running from Oakhill *via* Downside, the level crossing and Cannard's Grave to Prestleigh, excluding the said road.

In the Petty Sessional Division of Wells City and County: The parish of West Pennard.

In the Petty Sessional Division of Weston: The parishes of Newton St. Loe, Englishcombe and Dunkerton and so much of the parish of Camerton as lies to the north and west of the main road running from Radstock to Bath, excluding the said road.

In the Petty Sessional Division of Temple Cloud: The parish of Farmborough.

In the Petty Sessional Division of Keynsham: The parishes of Marksbury and Priston.

In the Petty Sessional Division of Weston: The parishes of Claverton and Bathampton and so much of the parishes of Bathford, Batheaston and Swainswick as lies to the south and east of the main road running from Box to Bath, including the said road.

IN THE COUNTY OF SOUTHAMPTON (HAMPSHIRE)

In the Petty Sessional Division of Ringwood and Fordingbridge: The parish of Martin and so much of the parish of Damerham as lies to the north and west of the road running from Cranborne *via* Damerham to Rockbourne, including the said road.

IN THE COUNTY OF DORSET

The Borough of Blandford Forum.

In the Petty Sessional Division of Blandford: The parishes of Tarrant Gunville, Pimperne, Tarrant Hinton, Tarrant Launceston, Tarrant Monkton, Tarrant Rawston, Tarrant Rushton, Langton Long Blandford, Chettle, Farnham, Sixpenny Handley and Pentridge.

In the Petty Sessional Division of Shaftesbury: The parish of Ashmore.

In the Petty Sessional Division of Wimborne: The parishes of Cranborne, Wimborne St. Giles, Gussage All Saints, Gussage St. Michael, Long Crichel, Moor Crichel, Witchampton, Chalbury and Edmondsham and so much of the parishes of Horton and Woodlands as lies to the north and west of the road running from Chalbury *via* Horton and Woodlands to Edmondsham, including the said road.

IN THE COUNTY OF LONDON

The Royal Borough of Kensington.

The Metropolitan Boroughs of Hammersmith, Paddington, Hampstead, St. Marylebone, St. Pancras, Finsbury, Holborn, Shoreditch, Hackney, Stoke Newington and Islington.

IN THE COUNTY OF MIDDLESEX

The Boroughs of Acton, Ealing, Willesden, Wembley, Harrow, Hendon, Finchley, Hornsey, Tottenham, Edmonton, Southgate, Enfield and Wood Green.

The Petty Sessional Division of South Mimms.

In the Petty Sessional Division of Highgate: The parish of Friern Barnet.

In the Petty Sessional Division of Uxbridge: The parish of Ruislip.

IN THE COUNTY OF ESSEX

The Boroughs of Leyton, Walthamstow and Chingford.

IN THE COUNTY OF HERTFORD

The City of St. Albans.

The Borough of Watford.

The Petty Sessional Division of Barnet.

In the Petty Sessional Division of Cheshunt: The parish of Cheshunt.

In the Petty Sessional Division of Hatfield: The parishes of Northaw and North Mymms and so much of the parish of Bishop's Hatfield as lies to the south and east of the main road running from Hertford *via* Hatfield to St. Albans, including the said road.

In the Petty Sessional Division of St. Albans: The parishes of Colney Heath, London Colney and St. Stephen.

In the Petty Sessional Division of Watford: The parishes of Aldenham, Bushey and Watford Rural and so much of the parish of Abbots Langley as lies to the south and east of the main road running from St. Albans to Watford, including the said road.

In the Petty Sessional Division of Hertford and Ware: The parishes of Little Berkhampsted and Essendon.

IN THE COUNTY OF WILTS

The Borough of Devizes.

In the Petty Sessional Division of Devizes: The parishes of Stert, Potterne, Worton, Marston, Seend, Poulshot, Rowde, Roundway and Etchilhampton and so much of the parish of Bishops Cannings as lies to the south of the road running from Devizes *via* Horton to Allington, including the said road.

In the Petty Sessional Division of Devizes: The parishes of Allcannings, Stanton St. Bernard, Beechingstoke and Patney and so much of the parish of Bishops Cannings as lies to the north of the road running from Devizes *via* Horton to Allington, excluding the said road.

In the Petty Sessional Division of Everley and Pewsey: The parish of Woodborough and so much of the parish of North Newnton as lies to the west of the main road running from Upavon to Pewsey, excluding the said road.

In the Petty Sessional Division of Whorwellsdown: The parish of Bulkington.

In the Petty Sessional Division of Bradford: The parishes of Monkton Farleigh, Atworth and Broughton Gifford and so much of the parish of South Wraxall as lies to the north and west of the road running from Bradford-on-Avon *via* Bradford Leigh to Great Chalfield, excluding the said road.

In the Petty Sessional Division of Melksham: The parishes of Melksham Within and Melksham Without and the lands common to the parishes of Broughton Gifford and Melksham Without.

In the Petty Sessional Division of Chippenham: So much of the parish of Lacock as lies to the south and west of the road running from Melksham *via* Lacock to Corsham and so much of the parishes of Corsham and Box as lies to the south and east of the road running from Lacock *via* Corsham and Box to Bath, including the said roads.

In the Petty Sessional Division of Tisbury and Mere: The parishes of Donhead St. Mary, Donhead St. Andrew, Ansty, Alvediston, Berwick St. John and Tollard Royal.

In the Petty Sessional Division of Salisbury and Amesbury: The parishes of Ebbesborne Wake, Bower Chalke, Broad Chalke, Bishopstone and Stratford Tonay.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Dorset. Trapdoor Farm, East Orchard, Shaftesbury (Jan. 31).

Essex. Rayne Hall, Rayne, Braintree (Jan. 31).

Hereford. Aubreys Farm, Bromsborrow, Ledbury (Feb. 1).

Lanarks. Langdale Farm, Greengairs, Airdrie (Jan. 27).

Yorks. Rudby Farm, Hutton Rudby, Yarm (Jan. 29).

Fowl Pest

Hants. The Laurels, Canada Road, West Wellow, Romsey (Feb. 2).

Hunts. Meadway, Church Lane, Fenstanton, Huntingdon (Jan. 28).

Lancs. Cloyhern Nurseries, Carr Lane, Tarleton, Preston; Sydbrook Farm, Sydbrook Lane, Mawdesley, Ormskirk; Beechwood, Chainhouse Lane, Whitestake, Preston (Jan. 28); Wallflower Cottage, Sluice Lane, Rufford, Ormskirk; Fanny's Farm, Greenhalgh, Kirkham, Preston; South View, Flag Lane, Penwortham, Preston; Sinclair Farm, Weeton Road, Wesham, Preston (Jan. 29); Croftlands, Brown Edge, Bamber Bridge, Preston; 131, Mossy Lea Road, Wrightington, Wigan; 19, Pendle Street East, Sabden, Blackburn; 21, Pendle Street East, Sabden, Blackburn (Jan. 30); Green Lane Farm, Catforth, Preston; 42, Halsall Road, Birkdale, Southport; Sharrocks Farm, Green Lane, Catforth, Preston; Lingard Hall, Catforth, Preston (Feb. 2).

Norfolk. Poultry Farm, Sandy Lane, Taverham, Norwich (Jan. 28).

Yorks. Applegarth, Manor Farm, Wilbury, Northallerton; Chestnut View, Deighton, Northallerton (Feb. 1).

Swine Fever

Beds. Briggington House, Hockcliffe Road, Eggington, Leighton Buzzard (Jan. 28); Plots 227-228, Mile Road Allotments (Jan. 29); The Maltings, Ampthill Road (Jan. 30).

Birmingham. Bay Tree Bungalow, Hillwoode Road, Woodcock Lane, Northfield (Jan. 27).

Cumbs. Fernwood, Eden Hall, Penrith (Jan. 27).

Herts. The Piggeries, Smallholding, Cromer Hyde, Welwyn Garden City; Mill Farm, Charlton, Hitchin (Jan. 27); Woodlands Poultry Farm, Hyde Lane, Hemel Hempstead (Jan. 28).

Leics. Glebe Farm, Goadby, Billesden (Jan. 30).

Norfolk. Heywood Hall, Diss (Jan. 30); Poplar Farm, Roydon, Diss (Jan. 29).

Somerset. Bonnetts Farm, Trudox Hill, Frome (Jan. 31).

Suffolk. Mere Close Farm, Haughley, Stowmarket (Jan. 28); Hall Farm, Stanton, Bury St. Edmunds (Jan. 29); Pinnocks Farm, Barningham, Bury St. Edmunds (Jan. 30).

Surrey. Milford Chest Hospital, Milford, Godalming (Jan. 28); Place Farm, Park Road, Banstead (Jan. 29); Grove End Farm, 18, Yew Tree Way, Addington, Croydon (Jan. 31).

Wilts. Long Ground Farm, Bulkington, Devizes (Jan. 31).

Worcs. Brockhill Farm, Redditch (Jan. 30).

Yorks. Park Farm, Londesborough, Market Weighton (Jan. 31).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Methods of Castration

Sir,—I was most interested in the article in THE RECORD of February 1st entitled "The effects of different castration methods on the growth and well-being of calves." Fenton *et al* should be congratulated, as the collection of this sort of information, on such an everyday problem, is invaluable to the practising veterinary surgeon.

The authors have refrained from too much generalisation, but the "elastrator" method has not come out of the investigation unscathed. Since its introduction I have been unhappy with the overall results and in both calves and lambs have encountered considerable trouble. The initial pain following application can be severe and, I think, may last longer in some cases than this article suggests. The inflammatory reaction above the site, which follows later, can be marked and may cause unthriftiness, loss of weight and even death, and when castration by this method is incomplete the resulting inguinal testicle may go unnoticed until marked masculine behaviour is observed. In lambs the ulceration produced is an ideal site for tetanus bacilli to multiply and an alarming percentage of lambs can become infected. Only one such calf has died from tetanus in my practice, probably because of natural resistance or because in my area they are nearly always housed following operation.

I feel that this article has presented factual information on an important problem and "42 days after application there was some evidence of swelling and

pain in all calves" is rather damning evidence. This method should be discouraged.

Yours faithfully,
RICHARD H. C. PENNY.

The Black Lion Yard,
High Street,
Leighton Buzzard,
Beds.
February 4th, 1958.

Sir,—It may be useful to draw attention to the differences between lambs and calves in response to castration by various methods.

The work published in THE VETERINARY RECORD dated February 1st, 1958, by Fenton, Elliot and Campbell indicates a preference for the Burdizzo method as the one causing the least pain and having the minimum retarding effect on the progress of the calf. It is noted, however, that the differences in overall weight gain between the methods observed were not significant.

Investigations carried out at Leeds (Barrowman *et al.* 1953, 1954) with lambs, similarly failed to show a significant difference in live-weight gain, between groups castrated by the rubber-ring ligature and crushing techniques. Further, no difference resulted in the performance of lambs following castration with rubber-ring ligatures at different ages. The Burdizzo technique in lambs produced appreciable scrotal and testicular swelling which developed and persisted for a very much longer period than was the case with the calves recently reported. The rubber-ring technique, on the other hand, when employed within 7 days of birth for castration and within 48 hours of birth for tailing was found to cause considerably less discomfort.

Yours faithfully,
K. G. TOWERS.

Department of Agriculture,
The University,
Leeds, 2.
February 4th, 1958.

References

BARROWMAN, J. R., BOAZ, T. G., & TOWERS, K. G. (1953). *Emp. J. of Exp. Agric.* **21**. 193.
———, ———, & ———. (1954). *Ibid.* **22**. 189.

Metabolic Disorders in Hill Sheep

Sir,—I find the article by Messrs. Hughes and Kershaw on metabolic disorders in sheep intensely interesting and I should be glad to have a little further information.

It is stated that the lowland pasture in the first farm was fertilised by the drainings from the mixen. Had the lowland pastures on either of the farms already received early dressings of nitrogen in order to produce early grass, and what were the grasses which went to make up the herbage?

It has long been felt that weather changes in some way influence metabolic diseases; and I wonder if the difference in barometric pressure between the upland and lowland pasture could simulate the barometric change occurring under different weather conditions?

The almost immediate display of symptoms following the change seems too sudden to be associated with the grazing. With hypomagnesaemia in cattle there is generally a delay of a few days after moving onto new pasture before the onset of symptoms.

It would also be interesting to know just how close to lambing were these ewes; and whether the udders were already functional.

Yours faithfully,
J. B. WHITE.

The Spinney,
63, Bouverie Avenue,
Salisbury.

January 26th, 1958.

Sir,—May I refer to Messrs. L. E. Hughes's and G. F. Kershaw's interesting paper "Metabolic Disorders Associated with Movement of Hill Sheep." (*Vet. Rec.* **70**. 77.).

In this area, cattle are normally walked up to 16 miles to sales, and then a further 8 miles to the pier for shipment to the mainland. It is common to find several in-calf cows go down and present symptoms similar to those described in the paper. Treatment by injection with calcium effects a speedy recovery. Similar symptoms, shown in a small flock of Blackface ewes moved from one farm to another shortly before lambing in 1957, were quickly resolved by the same treatment.

Older shepherds have told me that these symptoms are common when in-lamb ewes are moved long distances, and are quite distinct from exhaustion.

It would seem to me that the condition is precipitated by the stress of forced walking after a quiet period of grazing, and not by any change in feeding.

Yours faithfully,
T. JARRETT.

Newton House,
Bridgend,
Isle of Islay.

January 27th, 1958.

The Feeding of Turkeys

Sir,—Your correspondent, Mr. T. Barton Mann, Director of Veterinary Research, W. A. Motley, Ltd., implies in his long and interesting letter in last week's VETERINARY RECORD that proprietary poultry feeds should be viewed with suspicion (or perhaps not used at all) because they may lead to increased mortality or morbidity. Needless to say this is a view which many of us do not share.

What particularly interests me is the fact that Mr. W. A. Motley himself, in a first-rate article in the current issue of *Turkeys*, fails apparently to take full notice of the advice offered by his own Laboratory and Research Division! If I were to quote various sentences from Mr. Motley's paper I should doubtless be accused of extracting particular parts for the benefit of my argument. If I may, Sir, I would like to

quote the whole of the section devoted by Mr. W. A. Motley to "Foods and Feeding," which reads:—

"In addition to trying several breeders' poults, my recommendation would be for growers to try several compounders' foods also. Even these may vary from season to season and a comparison can prove very illuminating.

"Deterioration in storage is often more rapid with modern compounds than with the more simple diet, and the grower should get first-hand advice from the compounders. Trace elements have a great deal to do with this. Read the printed instructions on the bags and follow the directions given. They are printed for a purpose. It really is amazing how oblivious some people are to this printed matter.

"If poults are being brooded in small lots of about 70 intensively in outdoor brooders with direct sun, a fast crumb without preventive drugs may be the best and the cheapest. If, on the other hand, the method of brooding is in large groups involving several breeders' poults in 'battery' or 'pole-house' conditions, where the poults see much less of the attendant, they may need foods which include the 'docility factor' plus 'preventive drugs' all adding to the expense.

"Crumbs are the fastest growing food, but it is not always the fastest that is required, particularly with 'about-the-end-of-April' hatched birds. Here a low protein diet and the birds on grass will be slower and much cheaper, but with June-hatched onwards the fastest foods and all birds intensively reared would be the order of the day if the Christmas market be aimed at. Damp mash is a help at any age if the extra time involved does not create undue expense.

"Do make an effort to rid the farm of rats and mice, and for that matter as many wild birds as possible; they all help to increase the risk of a 'flare-up' of disease.

"Be strict about keeping the food bags very dry. Mouldy food can cause trouble which is not easily cleared up. If any part of the food is ground by the grower, remember that ground wheat by itself will only keep for a matter of a few days."

Yours faithfully,
W. P. BLOUNT.

The British Oil and Cake Mills, Ltd.,
2, Kingscote Street,
London, E.C.4.

January 29th, 1958.

Promazine Hydrochloride in Canine Practice

Sir,—I have been using recently a new drug, which has proved of great value in canine work, and my findings might be of interest to others.

This compound, promazine hydrochloride,* is of the phenothiazine group, but differs from the more commonly used chlorpromazine hydrochloride in its lower irritability and toxicity (*J.A.V.M.A.*, 1957). It induces calmness and has been used in over 100 dogs with no side-effects other than the one example mentioned below. The indications for which I have found this drug useful are extremely varied and I am constantly discovering new ways in which it helps me to control and treat difficult dogs.

The main indication is the need for restraint, in all the forms in which it is required in canine practice. Prior dosage by the owner allows examination by the veterinary surgeon, especially of eyes, ears, teeth, and tonsils. Administration by owner or veterinary surgeon greatly facilitates clipping, grooming, dressing wounds and sores, post-traumatic suturing, and, most important, helps the dog to remain still while X-rays are being taken.

It has quietened kennel dogs, and was of great value in quietening other dogs during thunder and fireworks. It also permitted trouble-free travel, helped in the treatment of pruritus and emesis, and prevented licking of stitches. The drug was also used as a premedication for anaesthetics. Here it was usually given intravenously and, after the full effect had been achieved, the anaesthetic was given. It was found that only about half the usual dose of intravenous anaesthetic was needed.

Promazine was used in two main ways; it was given either by mouth, when the owner was administering it, or by injection when administered by myself. These injections were generally intravenous, which was more convenient, but the intramuscular route was used if an alternative was called for. Dosage was of the order of 1 to 2 mg. per lb. bodyweight when injections were used, and 2 mg. per lb. orally, although greater amounts were occasionally given.

Intravenous therapy produced an effect in 2 to 4 minutes, and intramuscular therapy in 15 to 20 minutes. There was no unusual pain at the injection site, no pallor of the mucous membranes was observed (Weaver, 1956), and, apart from a single case in which a greyhound showed transient twitching of the neck muscles after the rapid intravenous injection of 100 mg., no untoward side-effects have followed its use by that route.

In summary, my use of promazine hydrochloride in over 100 dogs has shown me its great value in producing restraint, in allowing difficult examinations, dressings, and suturings to be made, and in facilitating anaesthesia.

Yours faithfully,
F. G. GREER.

79, St. Helen's Road,
Leigh,
Lancs.

January 9th, 1958.

* Sparine, John Wyeth and Brother, Ltd.

References

J.A.V.M.A. (1957). *Editorial.* **165.** 1,619.
WEAVER, B. M. Q. (1956). **68.** 349.

Scientific Contributions as Letters

Sir,—I am a little surprised to see that you published in your issue of December 21st two letters in

the correspondence column which are scientific contributions and I wonder whether the implications of such a practice are fully appreciated by yourself and the Editorial Board. Both these letters are quite valuable scientific contributions and I should have thought would more properly have been published as such, perhaps enlarged slightly.

There are, I venture to suggest, two objections to publishing brief scientific communications as letters: (a) for the sake of brevity the authors are inclined to give bare results without the experimental detail, etc., necessary for other people either to judge the work or to attempt to repeat it; (b) the authors have an unfair advantage of very rapid publication which might give them priority over someone who conformed with the accepted scientific practice and published a properly prepared article with all the delay that entails both in writing and awaiting publication.

There is a great difference between the correspondence column in THE VETERINARY RECORD and "Letters to the Editor" in *Nature*. The latter is almost a unique feature in the scientific press. It does not, in fact, provide a medium for rapid publication as there is a delay of something like 4 months in contributions appearing under this heading. The correspondence columns of a professional journal are something quite different (could you imagine the correspondence that has been included in THE VETERINARY RECORD over the last six months or so appearing under "Letters to the Editor" in *Nature*?); "Letters to the Editor" is really a misnomer, but presumably has its origin far back in the past.

I concede that there might be special circumstances occasionally, for example when it is urgent that members of the profession should be given some information of immediate practical importance, or when there is an appeal for material for research purposes. I do not think this applies to either of the letters just published.

Yours faithfully,
W. I. B. BEVERIDGE.

Department of Animal Pathology,
School of Veterinary Medicine,
Madingley Road,
Cambridge.

January 3rd, 1958.

The "Space Dog"

Sir,—I have followed the correspondence on the "Space Dog" with interest and no little amusement. I did not enjoy the thought of what the unfortunate animal went through in pain and anxiety. I am not sadistic I assure you.

I was amused, however, at the thought of Mr. Hamilton Kirk, Mr. Wall, and Mr. Maguire sitting down to pen indignant letters in protest against the "diabolical" treatment meted out to this one dog. Their intentions were good, I readily admit, but were they, in fact, in any position to protest?

Presumably these gentlemen are practising veterinary surgeons and if asked to do so would "maim" dogs and bitches, toms and tabbies, at the request of owners.

No veterinarian in these days can afford not to make use of the various vaccines and sera at his disposal for the treatment or prevention of animal ailments. To do so would be, financially, suicidal and ethically, criminally negligent. Yet when making use of such vaccines, etc., I wonder if any of the above gentlemen ever pause to consider the amount of suffering associated with their discovery and everyday manufacture? I doubt it. If they do, however, they, being intelligent men, realise that some suffering is unavoidably demanded from a few for the salvation of many.

"Laika," poor animal, suffered, and most horribly so, but let us hope that some day her experiences will prove of inestimable value to mankind as a whole.

There are two courses open to Mr. Hamilton Kirk, Mr. Wall, and Mr. Maguire. Either become fanatical antivivisectionists with all that this implies, or, more rarely, keep some sense of proportion when scientific experiments involving animals are called for if mankind is to continue to progress.

Yours faithfully,
M. J. HENIGAN.

Straits Racing Association,
P.O. Box No. 3011,
Singapore.

January 26th, 1958.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN
Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-Mouth	Fowl Pest	Sheep Scab	Swine Fever
16th to 31st December, 1957		11	1	19	75	—	39
Corresponding period in	1956	15	—	7	72	—	23
	1955	17	1	2	137	—	31
	1954	25	—	—	38	—	68
1st January to 31st December, 1957		318	9	184	1,034	—	960
Corresponding period in	1956	1,245	11	162	956	—	741
	1955	764	8	9	906	—	1,403
	1954	350	4	12	795	—	1,455